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THE PAINFUL SHOULDER.[1]

By SELWYN NELSON,
Sydney.

PAINFUL disorders of the shoulder region are common. They may arise in three ways: (i) from disease of local structures; (ii) from lesions of the brachial plexus or its central connexions; (iii) from visceral pain referred to the shoulder.

In some cases the shoulder pain is the sole complaint, in others it forms part of a more widespread symptomatology. In monosymptomatic cases careful physical examination may reveal signs of involvement of the nervous system or of other parts of the locomotor system.

Local Lesions of the Shoulder Region.

The structures in the shoulder region which may become painful include the shoulder joint, the acromio-clavicular joint, the joint capsules, the subacromial bursa, the supraspinatus tendon and the long head of the biceps, and the muscular and fibrous tissue around the shoulder. Many of the painful affections of these structures are of the so-called "rheumatic" variety, rather indefinite in pathology and ætiology. Some are traumatic, some associated with degenerative changes. Osteoarthritis is extremely rare in the shoulder joint, and only slightly less uncommon in the acromio-clavicular joint. Rheumatoid arthritis frequently affects the shoulders as part of the development of the classical form of the disease, but in such cases is bilateral. If the patient complains of pain in the shoulder and no other joint is involved, rheumatoid arthritis is the last thing to think of in making a diagnosis. Capsulitis of the shoulder joint, subacromial bursitis, supraspinatus tendinitis with or without calcification, and tendinitis of the long head of the biceps are common causes of painful shoulder. The pain is of an aching character, localized to the shoulder region or referred to the outer aspect of the upper arm. The movement of raising the arm is painful and limited. Internal and external rotation are restricted. Local tenderness may be demonstrated over the lateral aspect of the shoulder just below the acromial shelf when the bursa or the supraspinatus tendon is involved, or anteriorly in cases involving the long head of the biceps. In the latter cases there is much less limitation of movement.

There is little tendency to spontaneous recovery. Restriction of activity of the joint is followed by wasting of the deltoid muscle and capsular changes, the so-called "frozen shoulder". At this stage there may be some decalcification of the head of the humerus.

Treatment by assisted active movements combined with short-wave diathermy frequently gives very satisfactory results. When the pain is the determining factor in producing muscle spasm, injection of the subacromial region with 1% "Novocain" solution followed by gentle exercise of the joint is frequently very helpful. The treatment may be given daily and continued for a week or more. Deep X-ray therapy is of assistance in cases in which pain persists in spite of more conservative measures. Orthopædic treatment, such as manipulation, immobilization in abduction and external rotation, may be considered in some cases. Both conservative and radical measures have their successes and their failures.

Fibrositis may affect the trapezius muscle or the rhomboids giving rise to shoulder pain. Palpation of the muscles will reveal trigger zones, pressure on which reproduces the pain complained of. These zones may feel indurated. Injection

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 26, 1952.

of three to five millilitres of 1% "Novocain" solution into these areas frequently gives great relief. Several zones may be treated at one time, but repeated injections are often necessary at intervals of a few days. Sodium salicylate or acetylsalicylic acid should be given in these cases. It is frequently found that such patients have some psychopathic features. Careful interrogation will reveal two factors, one a life situation causing stress, the other an abnormal reaction to the life situation. The former may occasionally be relieved either by the patient or relatives, or with the assistance of an almoner. The latter may be ameliorated by psychotherapy. The mechanism of these psychic factors in the causation of fibrositis is obscure.

Neurological Lesions.

The neurological causes of shoulder pain include lesions of the cord or roots of the second cervical to the first thoracic spinal segments, lesions of the brachial plexus or peripheral nerves, and the little understood "reflex sympathetic dystrophy". While there is usually some other change, such as anæsthesia, muscle atrophy or change in tendon reflexes, this may not be apparent unless searched for. Anatomical variations of the brachial plexus, such as pre-fixation or post-fixation, may be associated with painful lesions of the shoulder region by making the plexus more vulnerable to pressure. In pre-fixation the contribution of the fourth cervical segment is considerable and that of the first thoracic segment negligible; in post-fixation the fifth cervical segment is poorly represented and the second thoracic segment makes a large contribution to the plexus.

The following list shows the main causes of pain in the shoulder region of neurological origin:

1. Lesions within the spinal canal: syringomyelia, cervical cord tumour, pachymeningitis (hypertrophic), adhesive spinal arachnoiditis, radiculitis, *herpes zoster*.

2. Vertebral disorders causing pressure on the cord or nerve roots: injury, cervical disk lesions, osteoarthritis of the cervical part of the spine, tuberculosis of the cervical part of the spine, neoplasm (primary or secondary) of the cervical part of the spine.

3. Brachial plexus lesions: brachial neuritis, cervical rib and related pressure lesions, Pancoast tumour, acroparæsthesiæ.

4. Reflex sympathetic dystrophies: causalgia, "shoulder-hand syndrome".

Lesions within the Spinal Canal.

Syringomyelia may be unilateral at the outset, and pain may dominate the picture. The diagnostic features are sensory changes of the dissociated type (loss of pain and temperature sense, with retention of touch) and a lower motor neuron lesion usually involving the hands.

J.B., a married woman, aged forty-six years, reported to her doctor in July, 1951, complaining of pain and numbness in the right hand and arm for two months. There was some deep tenderness over the brachial plexus, and she was treated with sodium salicylate and the vitamin *B* group. A month later the pains were worse, and there was deep tenderness in the forearm. The numbness was lessened. A septic tooth was located and removed. X-ray examination of the cervical region in October, 1951, showed no evidence of cervical rib or any other abnormality. Diathermy treatment to the shoulder region gave no relief. At this stage she was referred for opinion. Additional features were revealed on further questioning. The sensory disturbance consisted of a feeling of coldness, together with a difficulty in recognizing temperature variations with the right hand. There was some hyperæsthesia in the right forearm. There was some pain in the right shoulder aggravated by sneezing and coughing. There was also some increase in sweating in the right hand. On examination, she had hypalgesia and thermo-anæsthesia of the right side in the fifth, sixth and seventh cervical segments without any motor changes. A right Horner's syndrome was present. Biceps, triceps and supinator jerks were absent. The left side was normal. Lumbar puncture showed a normal pressure with a good response to the Queckenstedt test; the findings of cytological examination and biochemical tests were normal. A diagnosis of syringomyelia was made and deep X-ray therapy commenced. She has had two courses of treatment, and when she was reexamined in June, 1952, no extension of the lesion could be demonstrated. Pain had almost disappeared.

Cervical cord tumours are characterized by root pain radiating to the dermatome involved. The shoulder region is frequently involved, as many of the dermatomes of the arm region have a linear representation over the shoulder and outer aspect of the upper part of the arm. In addition there is hyperæsthesia, possibly segmental sensory loss and a segmental lower motor neuron lesion. The ascending and descending tracts of the cord are involved to a greater or lesser extent, and the sensory and motor changes arising therefrom may be demonstrated. Lumbar puncture will reveal signs of block—increase in the protein content of the cerebro-spinal fluid associated with impairment of the Queckenstedt response. Myelography will demonstrate the site of the lesion.

Pachymeningitis of the hypertrophic type may affect the cervical region. It may be due to tuberculosis, but syphilis is the commonest cause. The clinical picture is difficult to differentiate from that of cervical cord tumour. The Wassermann reaction is positive in the syphilitic cases, and there may be signs of syphilis in other parts of the nervous system. Manometric block is present. The pain is frequently bilateral. The thickened bands of *dura mater* in due course compress the cord, and the signs of compression may dominate the clinical picture.

Adhesive spinal arachnoiditis produces a patchy distribution of lesions in the arachnoid and pia, with interference with the cord both by compression and by interference with the blood supply. Any region of the cord may bear the brunt of the disease, pain in the shoulder and arm being present in cervical involvement. In one case of mine that came to operation, there were multiple small cysts of loculated cerebro-spinal fluid causing compression of the cord.

Vertebral Disorders.

Cervical disk lesions cause either a central or, more commonly, a lateral protrusion of the disk. In the former case no compression of local nerve roots occurs, the neurological signs being due to compression of the cord. The common site of protrusion is between the fifth and sixth or sixth and seventh vertebral bodies. Root pain is found distributed to the shoulder and to the sixth or seventh dermatomes with sensory and motor lesions of this segmental distribution. The pain is aggravated by coughing, sneezing or straining and by movement of the neck, usually lateral movement towards the affected side.

Osteoarthritis of the cervical part of the spine may cause pain in the shoulder. The mere presence of radiographic changes in the bones of the neck should not be taken as proof of this relationship. One frequently sees advanced osteoarthritic lesions of the cervical region, with narrowing of disk space between the fifth and sixth, and sixth and seventh vertebral bodies, with no evidence of compression of nerve roots at all.

C.S., a married woman, aged forty-one years, illustrated the coexistence of cervical disk degeneration and osteoarthritis of the cervical part of the spine. She presented a very complex symptomatology. There was pain in the back of the right shoulder, numbness at times in the right arm, at other times paræsthesiæ. In addition there were painful cramps in the arms and legs at night. She also complained of sacral backache made worse by standing. She has had several dizzy spells over the past two years, and on one occasion lost consciousness. The attack commenced as a feeling of extreme weakness, and the mouth started to twitch prior to loss of consciousness. She had had three surgical operations, two of them sections and one laparotomy for hiatus hernia. There was some limitation of movement of the neck, but lateral movement did not precipitate any pain in the shoulder or arm. No changes could be demonstrated in the sensation of the arm, nor could wasting or muscular weakness be demonstrated. She had, however, been relieved previously by the wearing of a belt collar holding the neck in hyperextension. The cramps and the twitchings were thought to be due to hyperventilation alkalosis, and symptomatic relief was afforded by the administration of quinine sulphate in five-grain doses at night.

Generally the pain from intervertebral disk protrusions is associated with demonstrable wasting of some muscle groups, such as the triceps, and some patches of diminished sensation can be demonstrated. For this reason it is thought that the pain in this case is due to pressure on the nerve roots by osteophytes.

Other diseases of the cervical part of the spine, such as tuberculosis and neoplasm, may produce shoulder pain. The diagnosis is made by recognition of the radiographic changes. In secondary neoplasm other sites may be involved and the primary lesion may be known.

Brachial Plexus Lesions.

Brachial neuritis as an entity is becoming rarer as more exact diagnosis removes from the inconvenient grouping various specific syndromes. One of these is the syndrome associated either with a cervical rib or with some compression of the neuro-vascular structures at the superior thoracic outlet by the first rib. Not all patients showing radiographic evidence of a cervical rib suffer any effect from it; the rib is a congenital abnormality, and symptoms arise frequently quite late in life, if at all.

The development of symptoms in later life points to a muscular factor allowing pressure to occur in a region made vulnerable by the structural abnormality. The actual symptoms vary from case to case. Shoulder pain is more liable to be a feature when the hand symptoms are mainly on the radial side. In many cases the ulnar side of the hand shows the sensory changes.

Wasting of the intrinsic muscles of the hand and of the muscles of the thenar eminence (*opponens* and *adductor pollicis*) is usually accompanied by some vascular effect. The subclavian artery may be compressed when the arm is by the side. A perceptible weakening of the radial pulse may be produced when the shoulder girdle is forcibly depressed.

B.P., a married woman, aged thirty-five years, was first examined in December, 1952. She complained of aching in the neck and shoulder, worse on the right side for several months. She also suffered from pain over the top of the head, sacral backache, indigestion, shortness of breath, palpitation and a swollen feeling in the throat. She brought along X-ray pictures of the cervical part of the spine, which she said showed arthritis and a cervical rib. The cervical rib proved to be a small one on the left side, and the arthritic changes minimal. Examination of the patient showed an excitable woman with a normal cardio-vascular system. There were no neurological signs suggestive of pressure on any component of the brachial plexus. There were tender areas in both trapezius muscles, and some deep in the cervical region posteriorly. It was obvious that there was some deep emotional disturbance producing this symptomatology, and further questioning revealed many factors in her life situation, any of which could contribute to her state of health. She was married to a man thirteen years her senior, who was rather aloof. She had developed a nervous disorder after the birth of her second child six years before. For this she had shock treatment at Callan Park. Subsequently a hysterectomy was performed, and further nervous symptoms were treated by further electroconvulsive therapy. At the time of examination, her younger child was under treatment for rheumatic fever and she was very worried about this. Her husband, she alleged, treated her complaints with scorn and had very little sympathy with her efforts to obtain medical treatment. She has been reassured about her essential good health and treated by local "Novocain" infiltration into the trigger zones. These visits have been made the excuse for psychotherapy. At the same time short-wave diathermy has been used for the painful areas. Her progress has been very satisfactory, and she has lost not only most of the pain, but also the other psychoneurotic symptoms as well.

Involvement of the structures of the apex of the lung by neoplastic tissue, the so-called Pancoast tumour, will result in infiltration of the brachial plexus and the cervical sympathetic chain. In addition to shoulder pain there may be wasting of the small muscles of the hand and Horner's syndrome.

The condition known as acroparæsthesiæ is usually found in women about the time of the menopause. The chief complaint is an unpleasant tingling sensation noticed especially at night, affecting the hand, often associated with pain in the upper part of the arm and shoulder. It may be bilateral, but in my experience is much more commonly unilateral. In these unilateral cases the right side is usually affected in right-handed persons. No objective neurological signs can be demonstrated. In some cases there is a history of blanching or coldness of the hands, but one cannot demonstrate any weakening of the radial pulse on manipulation of the arm. Walshe has postulated that a sagging of the shoulder girdle muscles may cause traction and compression of the lower portion of the brachial plexus. Vascular changes may also play a part. Relief may be given by exercises designed to improve the tone of the shoulder girdle muscles combined with substitution therapy to correct any hormonal deficiency. The carrying of heavy shopping bags should be forbidden.

Reflex Sympathetic Dystrophy.

Painful lesions in the upper extremity may be due to a disturbance of the sensory mechanism which has been attributed to sympathetic dysfunction. Causalgia arises as a post-traumatic phenomenon, especially when there is an incomplete lesion of a nerve. Burning pain of agonizing degree occurs in association with vasomotor and trophic changes. The severity of the pain is such that the whole life of the patient is devoted to the care and protection of the afflicted extremity. This type of condition is but rarely seen in civilian practice. What has been described by Steinbrocher as the "shoulder-hand syndrome" is, however, a fairly common occurrence. The clinical picture described by him passed through three phases. The first was a painful disability of the shoulder associated with either sudden or insidious onset of generalized swelling and stiffness of the hand and fingers. The second phase, coming on three to six months after the onset of the first, and occupying a similar period of time, consisted of both gradual resolution of the shoulder condition and reduction of the swelling of the hand; stiffness and flexion deformity of the hand then became manifest. During the third stage trophic changes of the hand became noticeable.

Askey (1941) had already described a somewhat similar condition occurring after myocardial infarction, while Johnson (1943) had given an account of hand changes in the same condition. Young and Pearson (1952) have recently published a review of 23 cases in which the clinical features are described on a broader basis than in the other communications quoted. They list the causes as idiopathic, shoulder lesions, cervical disk disorder, coronary artery occlusion, hemiplegia, *herpes zoster*, occupational dermatitis.

The syndrome includes some or all of the following: stiff shoulder, thickening and contractures of the palmar fascia, stiffness of the fingers, vasomotor changes (vasodilatation followed by vasoconstriction), spotty or diffuse decalcification of bone.

There has been much speculation concerning the cause of the condition. The vasomotor changes appear to incriminate the autonomic nervous system. The changes in bone are similar to those seen in Sudeck's atrophy. This decalcification may be due to disuse or to an increased blood supply to bone, or to both. I have found that some relief follows the use of the ganglion-blocking drugs such as "Etamon". White and Smithwick summarize the arguments in favour of a special set of "nocifensor" nerves located in the posterior root system, as postulated by Lewis, which have a vasodilator action. Evans (1946), discussing reflex sympathetic dystrophy, favours Lorente de Nó's concept of the internuncial pool of neurons—a closed self-exciting chain superimposed on the segmental reflex arcs. The level of this pool may be as high as the thalamic region. The efferent discharges may spill over to the contralateral side. In the present state of our knowledge of the anatomy of the autonomic nervous system no theory explains completely the observed phenomena of the condition. Patients who have an unstable personality, or who prior to the onset of the condition have had a tendency to cold, sweaty hands, appear to develop more persistent disability than more normal types.

It is important to differentiate this condition from arthritis. The appearance of the hand has a superficial resemblance to rheumatoid arthritis, in which swelling and vasomotor changes may be prominent. Closer inspection will show that the distal portion of the fingers are affected in the shoulder-hand syndrome, whereas in rheumatoid arthritis the changes are localized in the vicinity of the metacarpo-phalangeal joints and the proximal interphalangeal joints. Elevation of the erythrocyte sedimentation rate is not a feature of the shoulder-hand syndrome.

C.McC., a married woman, aged sixty-two years, was first seen in January, 1952, having had a small fibrous nodule excised from the palm of the right hand a few weeks previously. She complained of inability to close the right hand. Abduction of both shoulders was restricted and both hands showed small areas of thickening of the palmar fascia. The hands were of a normal colour and very slightly puffy. She stated that she had had a "stroke" nine months previously, without loss of consciousness and without paralysis. At this time she was unable to walk straight and veered to the right. While awaiting admission to hospital she suffered a further episode of dizziness and difficulty in walking. She recovered rapidly from this without any permanent abnormal neurological signs. Treatment included daily intravenous injection for two weeks of two millilitres of "Etamon" and a longer course of short-wave diathermy to the shoulders and paraffin wax baths to the hands. When examined in June, 1952, she had slight restriction of extension of the fingers and a full range of movement in both shoulders.

A.K., a married man, aged sixty-two years, first noticed pins and needles along the dorsum of the right hand in September, 1951. There was also numbness around the right side of the mouth. This lasted about three weeks. Then he suffered a sudden attack of loss of power in the right arm with clumsiness of the fingers lasting for three months. In March, 1952, he noticed pain in the right shoulder with limitation of abduction. On examination in June, 1952, the shoulder on the right side was painful on movement, and abduction was restricted to 70°. Internal and external rotation were also limited, so that he could not get his hand behind the back or to the back of the neck. His blood pressure was 160 millimetres of mercury (systolic) and 80 millimetres (diastolic). The right corner of the mouth drooped, and he had weakness of the right *orbicularis oris* muscle. Muscle tone on the right side of the body was increased, as were the tendon reflexes, and the right plantar reflex was upwards. Hoffmann's sign was present on the right side. No sensory changes could be demonstrated. Apart from the clumsiness associated with the upper motor neuron lesion of the hand, no changes could be demonstrated. The neck movement was restricted. X-ray examination of the cervical part of the spine showed osteoarthritic changes. There was no cervical rib. The shoulder showed decalcification, but no other lesion.

This case may be classified as an incomplete "shoulder-hand" syndrome following hemiplegia, or probably more correctly as a capsulitis of the shoulder associated with the immobilization following the upper motor neuron lesion.

Visceral Pain Referred to the Shoulder.

Three conditions commonly found produce shoulder pain. Diaphragmatic pleurisy and gall-bladder disease through irritation of the phrenic nerve may cause pain in the right shoulder. The referred pain of *angina pectoris* is usually felt in the left shoulder, extending down to the left hand. Occasionally the right side is involved. The associated symptoms and signs of both these conditions are usually sufficiently obvious to leave little doubt as to the diagnosis. The shoulder-hand syndrome is not a complication of *angina pectoris*, but of myocardial infarction.

Summary.

The painful shoulder has been discussed as a problem in differential diagnosis. It is beyond the scope of this paper to discuss in detail the treatment of the various conditions mentioned. Some attention has been paid to the mechanisms involved in the production of the pain in the more obscure conditions.

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PAINFUL SHOULDER.[1]

By RICHARD HODGKINSON,

Sydney.

MAN'S upright posture has freed his hands to do what his brain devises. This has meant progress. Modern manipulations often demand an extended and abducted position of the arm, and the anatomical adaptation of the shoulder joint has not entirely kept pace with this.

Functional Anatomy.

Movement depends on the smooth working of muscles which act as balanced couples. The short rotator muscles, namely, the supraspinatus, infraspinatus, *teres minor* and subscapularis, we call the musculo-tendinous cuff; and they contract with the deltoid to give smooth abduction, keeping the head of the humerus against the glenoid whilst the deltoid supplies most of the power. Too great relative pull by the deltoid in early abduction will elevate the head of the humerus against the acromion process instead of producing abduction. This can occur also when the musculo-tendinous cuff is too weak.

The subacromial bursa helps the musculo-tendinous cuff to slide under the acromial process during abduction.

The supraspinatus muscle inserted into the superior facet of the greater tuberosity has the most responsible site of action in this movement. It is also the weakest member of the short rotator muscles.

It has been demonstrated in the living (De Palma, 1950) that during abduction and external rotation the bicipital sulcus slides along the long tendon of the biceps.

Degeneration.

The anatomical deficiency of the shoulder joint in relation to meeting its demands is shown by the gradual degeneration of the tendons and other related structures. These changes are almost universal in the later decades. They do not always disturb the function, nor do they always produce symptoms.

Examination of the changes found *post mortem* in symptomless lesions makes it easy to appreciate how puzzling is the diagnosis of the cause of pain in any one patient.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 26, 1952.

Codman (1934) helped clear the air when he drew attention to the importance of the supraspinatus tendon and the subacromial bursa in the production of shoulder pain, and now the clinical picture includes the following: (i) calcareous tendinitis, (ii) ruptures of the musculo-tendinous cuff, (iii) bicipital tendinitis, (iv) frozen shoulder.

Calcareous Tendinitis.

The occupation of those affected by calcareous tendinitis is usually of a sedentary type. Typists, barbers and surgeons keep their arms abducted for periods and are very susceptible.

Males are affected more frequently than females, and the highest incidence is in the fourth or fifth decade.

Acute trauma may precipitate the condition in patients in the younger age group.

Clinically it may be subacute, acute or chronic.

The subacute form is the commonest, beginning with pain over the front of the shoulder. This pain is phasic, beginning at 60° of abduction. There is a catch at this point. Movement is restricted by spasm, and muscle atrophy occurs later. The calcium is amorphous and is in the supraspinatus tendon at Codman's critical point, which is seen in the X-ray film when an antero-posterior view is taken in full external rotation. Views in full internal rotation and in mid position are necessary as other tendons are often involved.

The tendinous fibres shred, hæmorrhage occurs, and calcium is deposited in the degenerating fibres. This appears before pain, which is due to stretching of the sensitive floor of the subacromial bursa. The exacerbation at about 60° of abduction is due to increased tension as the inflamed spot passes under the coraco-acromial ligament of the acromion.

The acute form is ushered in suddenly with loss of movement and intense pain. The prognosis is very much better.

The chronic lesion causes a jab of pain, which occurs during phases of movement and which is normal. It may be present for years. Subacute exacerbations are common.

Treatment.

Heat, analgesia and rest are the backbone of treatment. Certain relief of tension, which causes the pain, can be achieved by incision. Most cases respond to conservative means. Other methods of relieving tension, such as needling, rupture by manipulation, deep X-ray therapy, are uncertain.

Rupture of Musculo-Tendinous Cuff.

As with all shoulder lesions the symptoms of rupture of the cuff and those of tendinitis are much the same. However, there are certain important differences.

The cause is trauma—that is definite. The severity of the injury diminishes with the age of the victim. He is a male in nine cases out of every ten, past middle age and a labourer.

Clinically pain follows the injury, but it may become greater later when tension increases in the bursa. The arm is immobilized by spasm, and there is tenderness over the tip of the shoulder; sometimes a depression can be felt.

When the lesion becomes less acute, movement returns, spasm is reduced and pain is phasic as in tendinitis.

The pathology is interesting, the tear being either complete or incomplete. We have seen that a complete tear is compatible with apparently normal function; after the initial spasm settles down, loss of function is due to unbalance of the muscle couple. The powerful deltoid of the labourer over-acts, pulling the head of the humerus away from the glenoid and up under the acromion. The normal humero-scapular rhythm is destroyed.

Codman thought that complete tears should be sutured as early as possible. He infiltrated the area with "Novocain" to relieve spasm and operated on those who were unable to abduct the arm.

MacLaughlin (1944) appreciated that even with complete tears recovery could occur, and he has figures to show that late repairs of those tears that did not recover by heat and rest and early carefully graduated mobilization gave good results from surgery after six or eight weeks of conservative treatment.

His approach was transacromial; he excised ragged edges, sutured the edge down to bone and removed the acromion.

Bicipital Tendinitis.

Surgeons have been aware of disabling tenosynovitis about the wrist and in the fingers for some years. The importance of a similar condition about the long tendon of the biceps as it lies in the bicipital groove has not been so generally appreciated.

De Palma (1950) has shown that the groove moves on the tendon during abduction, external rotation and hyperextension.

There are two main groups of cases in which symptoms and signs of this condition occur: (i) those preceded by trauma, (ii) those without trauma.

The first group usually occurs in males, about forty years of age; often they are apprehensive and poor physical specimens. The trauma results in fractures about the shoulder, destroying the regularity of the bicipital groove. Dislocation may be sufficient or even simple falls on the elbow according to the age of the patient.

More commonly the condition occurs without previous trauma, but this is usually in women of an average age greater than that of the first group.

Clinically patients in these two groups present with pain in the anterior aspect of the shoulder and in the insertion of the deltoid. There is a general restriction of movement with pain beyond a limited range.

Lippmann (1943) clinches the diagnosis by searching for tenderness present along the bicipital groove and becoming very acute when the tendon is twanged under the thumb with the elbow flexed to a right angle. This tenderness may be found by simply rolling the tendon under the finger in the groove.

The acute condition may settle with rest and heat and graduated movements within the range of tolerance. It may become static, but (and this is important in susceptible individuals), it may progress to almost complete fixation of the humero-scapular joint, to what is, in fact, a frozen shoulder.

Treatment in these latter cases consists of freeing the tendon from its intraarticular attachment and from the groove and attaching it to the tip of the coracoid process.

Frozen Shoulders.

Frozen shoulder is common. It rarely occurs before the age of forty years, and usually the patient is a woman. She often is nervous and unstable and suffers from some metabolic disease.

Duplay (1896) called this humero-scapular periarthritis, and it became a commonly used medical dustbin. Codman (1934) and Lippmann (1943) and others showed that in susceptible individuals it could develop after all of the three conditions just discussed. De Palma (1950) considers that bicipital tenosynovitis is the commonest single cause and advocates transplantation of the long tendon of the biceps when Lippmann's sign is present as a means of relieving pain and allowing mobilization and gradually increasing activity to reverse the process. He describes the pathology very fully. The soil must be suitable, with degenerative changes present, as they usually are in the later decades, and pain from calcareous tendinitis, tears of the musculo-tendinous cuff or a bicipital tenosynovitis; this results in inactivity, stasis, formation of sero-fibrinous exudate and of fibrin, and lastly gumming of the large muscle planes, intramuscular areas, capsule and folds of synovia which abound in the joint, with final complete rigidity and very often relief of pain.

In treatment we should seek a diagnosis of the cause of pain, which should be attacked, often surgically, as early mobilization is necessary to reverse the process.

It is obvious, however, that there are other causes of this condition which are not understood, and much work is still required.

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SOME CLINICAL OBSERVATIONS ON ACUTE INFECTIVE HEPATITIS.

By B. P. BILLINGTON,

Fellow in Gastro-Enterology, Royal Prince Alfred Hospital, Sydney.

MUCH of the literature about the clinical manifestations of infective hepatitis stems from experiences in World War II, during which large numbers of cases occurred amongst service personnel under epidemic circumstances. Less, however, has been written about the sporadic occurrence of the infection in the urban community, possibly because patients are usually treated in the home by the general practitioner. The object of this paper is to draw attention to some clinical features of the disease which are less usual, but not rare. Forty-three patients, diagnosed clinically as suffering from infective hepatitis, whose jaundice was of five days' duration or under on their admission to hospital, were examined in the public wards of the Royal Prince Alfred Hospital, Sydney, during the period from May, 1949, to September, 1951. All but six resided locally and primarily sought medical advice at the hospital.

Pain.

Transient soreness, dull aching, or fullness felt in the mid-epigastrium, the umbilical region or in the right hypochondrium in the late prodromal and early icteric stages may be prominent features (Gordon, 1943). These symptoms are ascribed to swelling of the liver cords with tension producing an effect in Glisson's capsule and its projections; they are reported to occur in 20% to 75% of cases, and were a presenting complaint in 10 of these 43.

However, the onset with acute severe pain, sometimes associated with rigidity of the abdominal wall both in the right hypochondrium and in the right iliac region, is reported as being not infrequent in some epidemics, so that a mistaken diagnosis of an acute "surgical abdomen" may be made—acute cholecystitis, acute appendicitis or even ruptured ulcer (Barker *et alii*, 1945). In one series of 151 patients (Hardy and Feemster, 1946), 17 had severe abdominal pain, of whom five had localized pain and guarding in the right iliac region, and three had undergone appendicectomy before the correct diagnosis became obvious. Severe pain has been reported as more common in cases of malignant hepatitis progressing to a fatal issue (Alsted, 1947). Six patients of this present series had severe pain as a presenting symptom and were admitted to surgical wards; none, however, could be considered as suffering from malignant hepatitis.

CASE I.—J.W., a female patient, aged eleven years, was admitted to hospital with a history of nausea and sharp central abdominal pain lasting one day, followed by two days with vomiting, and sharp constant pain in the right iliac region. On examination of the patient, pronounced tenderness and guarding were present in the right iliac region. The patient had a dirty tongue and halitosis. There was no fever. The liver was not palpable and examination of the urine revealed the presence of acetone only. An immediate appendicectomy was performed, but the appendix was not inflamed. Three days after operation, the patient being still afebrile, bile was detected in the urine and there was tender enlargement of the liver. Subsequently the patient developed the full clinical and biochemical picture of infective hepatitis. It was learned that the patient's two sisters, aged ten and eight years respectively, had developed jaundice on the patient's sixth post-operative day.

CASE II.—P.Z., a male patient, aged eight years, presented with generalized colicky abdominal pain centred round the umbilicus of two days' duration, with vomiting but without diarrhœa or fever; there was no icterus on clinical examination, but pronounced tenderness and guarding were present in the right iliac region, and a provisional diagnosis of acute appendicitis was made until bilirubin was detected in the urine. The following day, pain, tenderness and vomiting had disappeared, but icterus was obvious on clinical examination, the liver was tender and palpable, and the thymol flocculation test produced a "++++" result. The patient's brother, aged four years, was admitted to hospital two days later with jaundice.

Four adults in the early icteric phase of the disease presented with severe upper abdominal pain, tenderness and guarding in the right hypochondrium and slight icterus; a provisional diagnosis of acute cholecystitis was made in three cases, and in the fourth biliary colic, as the pain was colicky and umbilical in type. In all four cases the pain, although severe at its onset, was transient, and jaundice deepened after the patient's admission to hospital. One of these cases showed a further hazard in diagnosis, in that the biochemical tests suggested biliary obstruction.

CASE III.—L.C., a female patient, aged forty years, presented with a history of severe epigastric pain, boring in character, felt also in the right hypochondrium, and also as an intermittent sharp pain in the right shoulder tip, which had lasted some eight hours, and pale stools for three days, without previous dyspepsia or history of exposure. The liver was enlarged three centimetres below the right costal margin on the patient's admission to hospital, and tenderness was localized, so that Murphy's sign was present. Biochemical investigations of the blood revealed that the serum bilirubin content was 26 milligrammes *per centum*, the serum did not react to the colloidal gold test, and the serum alkaline phosphatase content was 88 King units. On these grounds a diagnosis of obstructive jaundice was made. At laparotomy no abnormality could be found, except that the liver was enlarged diffusely, and an operation liver biopsy revealed active hepatitis with small foci of necrosis, but no evidence of biliary obstruction. The patient's post-operative course was stormy, with tachycardia, fever, and deeper jaundice for some forty-eight hours, necessitating the use of continuous intravenous glucose therapy. Subsequently the patient made a complete clinical and biochemical recovery.

Neurological Manifestations.

Lucké (1944), describing the features in 196 cases of epidemic hepatitis which proceeded to a fatal issue, showed that in 53% clinical neurological phenomena of a wide variety were present, and that at autopsy, in the majority, only œdema of the brain and acute non-specific degeneration of the ganglion cells were present, but in 15% there was evidence of mild meningo-encephalitis, which differed considerably from the lesions seen in other virus diseases affecting the nervous system. He concluded that the clinical and pathological manifestations were the result chiefly of the loss of detoxifying function of the severely damaged liver. Stokes *et alii* (1945) describe, also in fatal cases, lesions characterized by small and massive cerebral hæmorrhages due to the hæmorrhagic tendency in liver failure. At this hospital only one case has been seen since 1946, not included in this series, in which the acute infection proceeded rapidly to hepatic necrosis and death, with coma and generalized epileptic convulsions. At autopsy only some cerebral œdema was noted.

In cases which run a benign hepatic course, neurological complications may be seen and may arise either in the prodromal or less commonly in the early icteric phase of the disease. These are to be differentiated from the non-specific toxic delirium and semi-comatose state found in severe cases, often associated with dehydration due to vomiting—seen in two adults and one child of this present series. They include peripheral neuritis and acute symmetrical polyneuritis, with cytoprotein dissociation, which may commence in the prodromal phase or even up to the fourth week (Parker and Adams, 1947; Zimmermann *et alii*, 1947). Lescher (1944), however, describes a meningitic syndrome as the most common form, characterized by an increase in the pressure of the cerebro-spinal fluid, with increase in its lymphocyte and protein content, which may occur either in the prodromal phase or at the onset of jaundice. An encephalitic type, commencing in the prodromal phase, may also occur. He emphasizes that all such neurological complications have a benign prognosis; that the meningitic and encephalitic forms are usually transient and that the peripheral neuritis invariably clears completely, although weakness commencing in the prodromal phase may be present, but resolves three months after jaundice has disappeared. Both he and Brain (1943) record cases in which the onset of infective hepatitis with recovery was with convulsions and hemiplegia in the pre-icteric phase. It is suggested that the hepatitis virus in these cases may have "neurotropic properties" or that lowered resistance is a predisposing factor to neurological involvement by the hepatitis or other virus, as witness the occurrence of *herpes simplex labialis* in some cases of infective hepatitis. In this present series two patients showed transient neurological phenomena similar to those described previously, both in the prodromal phase of the disease.

CASE IV.—J.M., a male patient, aged eight years, presented with a history of malaise, headache and vomiting of one day's duration. On examination of the patient the temperature was 103° F. and tachycardia was present. The child appeared sick and was confused and lethargic. Neck stiffness was present, although Kernig's sign was absent. No other abnormality was noted. The following day, drowsiness, vomiting, photophobia, neck stiffness and a rise of temperature to 102° F. were present. At lumbar puncture the specimen of cerebro-spinal fluid was grossly contaminated with traumatic blood. Subsequently the boy improved rapidly and became afebrile, although malaise and anorexia remained. On the seventh day after his admission to hospital bile was noted in the urine, and the further progress was that of infective hepatitis.

CASE V.—A.A., a male patient, aged twenty-six years, was admitted to hospital in coma. Investigation of the history revealed that on the previous day the patient had complained of a headache and had been found unconscious two hours later. Subsequently the temperature had risen to 104° F. and rigors had occurred. On examination of the patient, his temperature was 102° F., there were athetoid movements of both upper and lower extremities and abnormal movements of eyes and jaws, and the pupils were widely dilated and inactive. There was left-sided facial paralysis, and neck stiffness was present, but Kernig's sign was absent; the deep reflexes were normal, the abdominal reflexes were absent, but the plantar responses were flexor in type; the patient had retention of urine, the only abnormal constituent of which was sugar. Small petechiæ were present on the neck and abdominal wall, and *herpes labialis* was found. Lumbar puncture showed the cerebro-spinal fluid not to be under increased pressure; it contained 40 milligrammes *per centum* of protein and 20 cells per cubic millimetre, 90% being lymphocytes. On the succeeding days the patient's condition gradually improved, the abdominal and pupillary reflexes returning on the third day after his admission to hospital, and a normal conscious state was regained by the fifth day. On the sixth day bile was noted in the urine for the first time, and on the subsequent day icterus was present and the liver was tender and enlarged. The subsequent course was that of infective hepatitis with recovery. On two occasions the Paul-Bunnell test produced a result within normal limits.

Cases IV and V represent the meningitic and encephalitic form of neurological involvement in the prodromal phase of infective hepatitis, the clinical manifestations in both commencing seven days before the onset of jaundice.

Hæmorrhagic Phenomena.

The occurrence of spontaneous bleeding in both acute yellow atrophy and chronic liver disease has long been known. With regard to acute infective hepatitis, Lucké (1944) noted the presence of macroscopic hæmorrhages at autopsy in 109 of 125 cases of acute fulminant epidemic hepatitis with a fatal issue. However, in the acute non-fulminant cases with a benign course, which constitute by far the majority of cases of infective hepatitis, Lichtman (1949*a*) states that few patients exhibit any particular hæmorrhagic tendency. Findlay *et alii* (1944), on the contrary, record 11 cases with bleeding in 432 cases of hepatitis following yellow fever inoculation. Yet in the present small series of 43 cases, in no less than six were multiple bleeding sites present, and in three hæmorrhage was of sufficient degree to warrant blood transfusion. There were large cutaneous ecchymoses in four cases, hæmatemesis and melæna in four, hæmaturia in three, and epistaxis, hæmoptysis, bleeding from the gums and bleeding *per vaginam* each occurred in one case.

CASE VI.—L.P., a male patient, aged twenty-one years, gave a history of illness of two weeks' duration, commencing with general malaise and anorexia followed by nausea and vomiting. Jaundice had been present and increasing in depth for three days. One of his workmates had been forced to give up work three weeks previously owing to similar symptoms followed by jaundice. On the day of his admission to hospital the patient had noticed a spontaneous bruise

over his right shoulder, and had four attacks of typical renal colic.

On examination of the patient his temperature was 100° F., and he presented some evidence of dehydration. Over the right shoulder was a large, fresh, continuous ecchymosis extending from the level of insertion of the deltoid below to the root of the neck above, from the ridge of the scapula posteriorly to within an inch of the nipple anteriorly. There were no other ecchymoses or purpuric spots. The liver was diffusely enlarged some three inches below the right costal margin; it appeared smooth and was tender. The urine appeared dark and smoky, ward testing showed acetone, bile and blood to be present, and profuse red cells were seen on microscopic examination. Laboratory investigations showed the findings typical of infective hepatitis, and the prothrombin index was estimated at 8%. Soon after his admission to hospital the patient had further hæmaturia, and then a large hæmatemesis followed by melæna. Vitamin *K* was given parenterally. Thirty-six hours later there had been no further bleeding or pain, and the prothrombin index was estimated at 97%, only 25 milligrammes of vitamin *K* having been given. It was considered on clinical grounds that blood replacement was indicated, and two pints were given. The patient subsequently made a rapid clinical and biochemical recovery.

The mechanism of bleeding in this case was probably the failure of the damaged liver to produce sufficient prothrombin for blood coagulation. The bleeding in four other cases is considered to be due to this mechanism, although the prothrombin index was not estimated before the commencement of parenteral therapy with vitamin *K*; yet at the time of estimation it was below 60% in all. One point of interest arises, which is well manifest by this case—namely, the fact that vitamin *K* administered parenterally can be readily utilized by the liver to form prothrombin in benign acute infective hepatitis without resort to blood transfusion to control bleeding rapidly. It was formerly held that the prothrombin response to parenterally administered vitamin *K* was a useful test of liver function and could be used to differentiate hepatocellular jaundice from extrahepatic obstructive jaundice; in the latter instance, in which the abnormality is failure in absorption of the vitamin, the response is excellent, but in the former failure of liver cell function prevents the utilization of vitamin *K* and so the response was alleged to be poor. Whereas, in the light of these cases, this cannot be held for acute infective hepatitis, it is certainly not uncommon in cases of chronic hepatic disease with jaundice, in which aspiration liver biopsy is required, for a week's parenteral therapy with vitamin *K* to affect a lowered prothrombin index very little. The rapid rise in prothrombin level in acute infective hepatitis was seen in another case of the series without bleeding, in which a prothrombin index of 64% on the patient's admission to hospital had risen to 100% after two days' treatment with vitamin *K* at the dose of five milligrammes a day. These rapid responses must surely represent rapid recovery in liver function, at least as regards prothrombin synthesis.

The sixth case of acute infective hepatitis with bleeding shows somewhat similar clinical features; but the cause of bleeding was not temporary prothrombin deficiency.

CASE VII.—E.G., a male patient, aged forty-four years, was admitted to hospital after a week's illness with anorexia and malaise; jaundice had commenced two days prior to his admission and had increased in depth. At the onset of jaundice there had been a transient episode, lasting twenty minutes, of stabbing pain in the right hypochondrium, which had been succeeded by vomiting, but no further pain. The patient had continued to vomit, and later vomitus contained old and bright blood. Two tarry stools had been passed. On examination of the patient he had no fever, but he was jaundiced; multiple small petechiæ were present over the skin of all four limbs and the trunk, with several larger ecchymoses. The liver was diffusely enlarged and tender. After his admission to hospital he had further hæmatemesis and melæna, with recurrent epistaxis. His hæmoglobin content was estimated at 6·7 grammes *per centum* and there was no leucocytosis. The prothrombin index was 100%, but there was abnormal capillary fragility, and the platelets numbered 40,000 per cubic millimetre. Liver function test results were consistent with a diagnosis of infective hepatitis. After blood transfusion the patient made a slow recovery; Graham's test produced a normal result, and liver biopsy revealed resolving hepatitis.

This patient presented with deepening jaundice and massive bleeding. The bleeding in this case was due not to prothrombin deficiency, but apparently to temporarily abnormal capillary fragility and deficiency in platelets. Cases of acute infective hepatitis in which there is such a degree of bleeding from this cause are uncommon in the literature (Alt and Swank, 1937; Jones and Evans, 1951); nevertheless it has been pointed out by Whitesell and Snell (1949) that thrombocyte and capillary defects are common in parenchymal disease of the liver, although there may be no clinical evidence of bleeding. These authors, dealing mainly with cirrhotics, showed in addition that in ten consecutive cases of acute hepatitis (cause not stated) five patients had significantly lowered platelet counts and seven gave an abnormal result to the capillary fragility test. On the other hand, in infective hepatitis the occurrence of occasional skin petechiæ alone without gross bleeding is well recorded (Lichtman, 1949*b*), and in this series it occurred in three cases, other than those in which gross bleeding occurred. The cause has not been elucidated.

This present series of cases appears to suggest that spontaneous bleeding and purpura in acute infective hepatitis are not so uncommon as previous reports would indicate. In some of the cases the bleeding was severe, with significant anæmia as a result, but in none was the hepatitis to be classified as fulminating, and complete recovery ensued in all; it would appear that spontaneous bleeding occurring in cases of acute infective hepatitis should not necessarily imply acute yellow atrophy and a grave prognosis.

Other Observations.

Of the 43 patients in this series, one presented with an initial story of polyarthralgia in the prodromal phase, and five were noted to have a fading punctate or blotchy erythematous rash on their admission to hospital. None had splenomegaly or lymphadenopathy. All these manifestations are recognized as occurring in acute infective hepatitis.

Three patients did not have obvious clinical jaundice at any stage of the disease, and diagnosis was made on the presence of a tender, enlarged liver, of bile in the urine, or of a rise in the serum bilirubin level with abnormal thymol flocculation. It is interesting to note that in many of the icteric cases the patients who were admitted to hospital appeared at the hospital only because the onset of jaundice had caused concern, whilst the malaise, anorexia and nausea which preceded it had not been considered of sufficient significance to warrant medical advice. Under these circumstances the current view that many sporadic cases of subclinical infection may occur without the patients' seeking medical advice seems probable, especially in the urban community, in which the heaviest incidence is in the earlier age groups, among whom anorexia, nausea and malaise may often be non-specific symptoms.

It is interesting to note that of the 43 patients only nine had a definite history of contact with a patient suffering from jaundice, and of these, six had their contact within the same home. The others, constituting almost four-fifths of the total, had, after specific interrogation, no known contact with another person suffering from jaundice.

Diagnosis.

CASE VIII.—A.L., a male patient, aged twenty-one years, was admitted to hospital with a temperature of 104° F. and a history of malaise, dry cough and pleuritic chest pain of two days' duration. On examination of the patient there were signs of bronchitis, and X-ray examination of the chest revealed no significant abnormality. After his admission to hospital his symptoms and fever subsided rapidly; but jaundice and a tender, enlarged liver were noted on the fourth day, and on the fifth a transient, blotchy, erythematous rash. A blood count showed the leucocytes to number 5850 per cubic millimetre, of which 36% were abnormal mononuclear cells; a Paul-Bunnell test was suggested by the hæmatologist to confirm a diagnosis of infectious mononucleosis; a positive titre of 1 in 10 only was obtained. Subsequently the patient made a satisfactory recovery, two further Paul-Bunnell tests being performed without significant change in the titre.

In this case, in which the clinical diagnosis of infective hepatitis was made, there was a significant number of abnormal mononuclear leucocytes in the peripheral blood; a Paul-Bunnell test was requested to exclude the possibility that the hepatitis might be a manifestation of infectious mononucleosis. It is well recognized that these cells may be seen in the peripheral blood in both conditions, and although the clinical diagnosis between the two is usually straightforward, generalized lymphadenopathy and splenomegaly can occur as manifestations of infective hepatitis, and similar types of neurological phenomena may be seen in both. A repeatedly negative response to the Paul-Bunnell test does not rule out the diagnosis of infectious mononucleosis (Kaufmann, 1944), and a significantly high titre may be found in acute infective hepatitis (Eaton *et alii*, 1944); skin rashes and abnormal responses to serum flocculation tests are found in both. What diagnosis, therefore, does one give in a case characterized by hepatitis, abnormal mononuclear cells in the peripheral blood and meningo-encephalitis, with a negative response to the Paul-Bunnell test similar to that described by Sherwood (1951)?

Although the diagnosis of acute infective virus hepatitis may be relatively easy under epidemic circumstances, when sporadic cases of hepatitis occur the affirmation that they are due to an infective virus may be difficult in the absence of a clinical means of implicating the virus.

CASE IX.—M.H., a *multipara*, aged twenty years, was admitted to hospital when three months pregnant. Her illness had commenced three weeks previously with recurrent pyelitis, which responded to sulphonamides. Anorexia persisted and later vomiting appeared. On the twelfth day of the illness jaundice appeared and subsequently increased in intensity. On the eighteenth day purpuric spots were seen on the abdomen and breasts, and later hæmaturia, hæmatemesis and melæna followed. Vitamin *K* was administered parenterally. There was no history of taking abortifacients or of previous dyspepsia, jaundice or abdominal pain. Examination of the patient on her admission to hospital revealed jaundice, skin petechiæ and ecchymoses, hæmaturia, and tenderness and enlargement of the spleen without fever; the hæmoglobin value was estimated at 8·4 grammes *per centum*, the prothrombin index was 50%, and the leucocyte and platelet counts were normal. A provisional diagnosis of infective hepatitis with bleeding was made, although both the thymol flocculation and alkaline phosphatase tests gave normal reactions. With blood transfusion and parenteral vitamin *K* therapy the acute manifestations subsided. However, icterus persisted, varying slightly in intensity, for a further two months, without fever but with leucocytosis. Liver function tests showed no significant change. During the second month the patient complained, for the first time, of attacks of sharp right upper abdominal pain; laparotomy revealed a solitary gall-stone impacted in the cystic duct. After cholecystectomy the patient made a complete recovery and she was delivered of a full-term infant three months later.

In this case of acute hepatitis, with recovery, the history of gradual onset of malaise and anorexia, followed by jaundice and bleeding due to a lowered blood prothrombin level without pain, fever or rigors, suggested the diagnosis of sporadic acute infective hepatitis. On the other hand, the jaundice persisted for nearly three months and fluctuated slightly, right upper abdominal pain in attacks appeared later, and there was persistent leucocytosis (but without fever). At laparotomy an abnormal gall-bladder with a large stone in the cystic duct was found; this suggested that the whole hepatic disorder was in the nature of ascending cholangio-hepatitis.

In a case of acute hepatitis with recovery the diagnosis that it is due to sporadic virus infection must be made rather by exclusion. It is important to exclude contact with known liver poisons, other infections including inoculation hepatitis, and the possibility of ascending cholangio-hepatitis. The so-called "typical history" of virus hepatitis is for the most part the history of a subacute onset of liver failure in miniature without specific features, mainly anorexia and malaise followed by jaundice. Patients with hepatitis due to arsenic or gold appear to give the same history unless the dose has been sufficiently large to induce rapid necrosis, and patients with chronic cirrhosis or established nodular hyperplasia appear to give a similar history when entering a decompensated phase. The pain and bleeding mentioned previously must surely be due to the hepatitis rather than specific for the virus infection. Liver function tests merely reveal that the liver is damaged, and liver biopsy may show that hepatitis is present with a certain architectural distribution without necessarily any demonstration of the ætiological agent. Of specific features, those which seem to point directly to a virus infection in non-fulminating cases are the presence of abnormal mononuclear cells in the peripheral blood, the presence of certain neurological phenomena, and possibly splenomegaly, but all these are uncommon. Perhaps the most common feature which may point to an infective virus as being the causative agent in any one case is the presence of a history of contact, which may be obvious under epidemic circumstances, but in this series of sporadic cases was present in only approximately one-fifth.

The importance of an accurate diagnosis of infective hepatitis is perhaps more obvious in retrospect. In general there are two schools of opinion as regards the relation of infective hepatitis to cirrhosis of the liver; (i) those who believe that infective hepatitis rarely proceeds to hepatic cirrhosis; (ii) those who take a different view, that in many cirrhotics the lesion may have resulted from an overt or subclinical attack of infective hepatitis at some time previously without full recovery. The history of an attack of jaundice some years previously, in a patient with obvious hepatic cirrhosis, does not necessarily imply that infective hepatitis was the precursor of the cirrhosis, nor that the prior attack of jaundice was not indeed due to the cirrhosis itself in a more active phase at that time. Even from the other end of the scale, when one is following a patient who has had a recent attack of hepatitis and who now has residual liver damage, rarely can it be definitely proven that, in fact, the hepatitis is due to a virus infection—except when the presence of a history of contact makes it extremely probable. A history of contact should therefore be sought, whenever possible, in the making of the diagnosis of sporadic acute infective hepatitis, and under present circumstances, in the absence of some other means of implicating the virus as the causative agent, the diagnosis must remain clinical. To elucidate the problem of infective hepatitis in relation to cirrhosis of the liver, further means are required to establish a definite diagnosis of virus hepatitis.

Summary.

1. Forty-three cases of acute infective hepatitis are reviewed.

2. In this series, phenomena which are described elsewhere as uncommon appeared not infrequently—namely, severe pain, neurological manifestations, and multiple bleeding sites.

3. Spontaneous bleeding in acute infective hepatitis may be quite severe and warrant blood transfusion.

4. The possible mechanisms of causation of bleeding are mentioned.

5. Some difficulties in the diagnosis of sporadic acute infective hepatitis are discussed.

6. The importance of attempting to obtain a history of contact is stressed in relation to the possible sequelæ of the disease.

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B.C.G. VACCINATION IN TASMANIA.

By G. SIBTHORPE,
Launceston.

SIX HUNDRED AND THIRTY-SEVEN people have been vaccinated from January, 1949, to December, 1951, at the Launceston Chest Clinic, Tasmania.

At the end of December, 1951, 534 had been retested two months after vaccination, and 207 of those vaccinated in 1949 and 115 of those vaccinated in 1950 were retested in 1951.

Until July, 1951, the reaction was examined one month after vaccination and the Mantoux test performed two months later. Since then the examination at the end of one month has been omitted, partly because I have gained the impression that it frequently takes longer than a month for a satisfactory reaction to develop, and partly to save an extra visit to the clinic.

The Mantoux test is carried out with 10 units of old tuberculin. The tests are performed by the clinic sisters. I check all the results myself, preferably after seventy-two hours, although sometimes circumstances necessitate the patients' being examined after forty-eight hours.

Those vaccinated consist of contacts of notified tuberculous patients, nurses, clerical and X-ray staff of the Launceston General Hospital, and a small number of the general public who have requested vaccination.

Of the 534 retested, 55·7% were nurses. The ages and the distribution of the 534 are as follows: 0 to 15 years, 181; 16 to 25 years, 306; 26 to 35 years, 29; 36 to 45 years, 13; over 45 years, 5.

Reaction to vaccination was analysed in order to determine how much batch-to-batch variation occurred.

For convenience all areas of infiltration were classified as papules 0 to 5·0 millimetres in diameter and papules over 5·0 millimetres in diameter. Any reaction with necrosis was classified as a pustule.

Tables I, II and III show the batch-to-batch variations for 1949, 1950 and 1951 respectively. These figures include 35 subjects who were vaccinated in 1949 and 1950, and who

TABLE I.

Month.	Batch Number.	Number Vaccinated.	Number Checked.	Papule 0 to 5 Millimetres.	Pustule or Papule Over 5 Millimetres.	No Reaction.
1949—						
January ..	42	12	11	6	3	2
March	50A	11	11	7	4	—
April	50	12	12	4	7	1
April	53	8	8	3	3	2
May	56	14	13	8	5	—
May	57	14	14	7	7	—
May	58	16	16	9	7	—
May	59	16	16	7	9	—
June	60	13	13	7	6	—
June	62	11	11	6	5	—
July	64	20	20	3	17	—
July	66	14	14	8	6	—
July	68	15	15	1	14	—
August ..	69	24	24	16	8	—
August ..	70	12	12	3	9	—
August ..	71	11	11	6	5	—
September ..	73	15	15	10	4	1
September ..	74A	7	7	6	1	—
September ..	75	5	5	2	3	—
September ..	76	8	8	4	4	—
October ..	78	8	8	3	5	—
October ..	79	6	6	2	4	—
November ..	83	6	5	5	—	—
December ..	86	2	2	2	—	—
December ..	88	4	4	4	—	—

were revaccinated in 1951 on account of reversion of the tuberculin response to negative.

There are considerable variations in the results. Apart from variations due to the vaccine itself, there are two factors which may be of importance in the causation of these variations.

TABLE II.

Month.	Batch Number.	Number Vaccinated.	Number Checked.	Papule 0 to 5 Millimetres.	Pustule or Papule Over 5 Millimetres.	No Reaction.
1950—						
February ..	97	9	9	8	—	1
March	99	5	5	1	—	4
April	106	11	10	1	9	—
May	110	2	2	1	—	1
June	112	5	4	2	2	—
June	115	9	9	7	—	—
July	117	14	13	13	—	—
July	118	5	5	5	—	—
July	120	3	2	2	—	—
August ..	122	7	7	3	4	—
September ..	125	4	4	4	—	—
September ..	126	21	20	17	3	—
October ..	131	14	14	13	1	—
November ..	134	3	3	3	—	—
November ..	135	6	5	5	—	—
November ..	137	4	4	3	1	—
December ..	139	8	8	5	3	—
December ..	140	8	8	4	4	—

The first is technique. Until October, 1949, all the vaccinations were performed by myself. During 1950 there were several changes of vaccinators, and since June, 1951, I have again performed all the vaccinations. Ustvedt quotes a report by Dr. Sophus Johansen of work in which four different vaccinators produced 98·1%, 91·4%, 95·6% and 93·1% of positive reactors in similar selected groups.

The second factor is the method of transporting the vaccine. It is packed in wood wool and flown to Hobart on Tuesday. On arrival it is put in a refrigerator, and on

Thursday it is sent by bus to Launceston (a journey of some four hours) and then placed in a refrigerator until required for use on Friday morning. It is possible that this period of exposure to room temperature may adversely affect the vaccine.

Dr. Triantaphyllou, in his report on B.C.G. vaccination in Greece, gives the following figures for vaccination at different months: May, 1948, 66·3% reactors; June, 1948, 47% reactors; October, 1948, 83·9% reactors; November, 1948, 89·4% reactors.

TABLE III.

Month.	Batch Number.	Number Vaccinated.	Number Checked.	Papule 0 to 5 Millimetres.	Pustule or Papule Over 5 Millimetres.	No Reaction.
1951—						
February ..	150	26	24	14	10	—
April	157	9	8	7	1	—
April	159	9	9	7	2	—
May	161	6	4	2	—	—
May	163	11	9	8	1	—
June	165	7	6	2	3	1
June	167	4	4	2	2	—
June	168	6	5	1	4	—
July	169	8	6	4	2	—
July	170	13	10	2	8	—
July	171	5	5	—	5	—
July	172	15	15	6	9	—
August ..	173	5	5	2	3	—
August ..	174	110	10	9	1	—
August ..	175	6	6	3	3	—
August ..	176	5	5	2	3	—
August ..	177	7	6	2	2	2
September ..	178	7	5	1	4	—
September ..	179	15	13	5	8	—
September ..	180	4	2	—	2	—
September ..	181	7	7	—	7	—
October ..	182	16	14	4	10	—
October ..	183	7	5	1	4	—
October ..	184	3	2	—	2	—
October ..	185	11	7	3	4	—
November ..	186	9	7	—	7	—
November ..	187	19	10	1	9	—
November ..	188	7	6	—	6	—
November ..	189	6	6	1	5	—

An analysis of the months in which vaccination was performed on those who had reverted to a negative tuberculin response one and two years after vaccination in 1949 and 1950 respectively was as follows:

(i) Subjects vaccinated successfully in 1949 and giving a negative response in 1951: January, one; March, one; May, seven; June, two; July, eleven; August, six; September, one.

(ii) Subjects vaccinated successfully in 1950 and giving a negative response in 1951: February, one; March, one; April, one; June, one; November, one; December, three. These figures do not point to a lower percentage of successful vaccinations in the summer, but are too small to be of significance.

It is interesting to note a description of a special refrigerated chamber for transporting the vaccine in the Second Annual Report of the International Tuberculosis Campaign.

Table IV shows the percentage of positive reactors two months and one and two years after vaccination. It shows quite a considerable fall in the percentage of reactors two years after vaccination. The lower percentage of positive reactors one year after vaccination in 1950 reflects the lower number of satisfactory reactions to vaccination produced during that year, although the difference cannot be shown to be statistically significant.

The Criteria for Successful Vaccination.

It is most important to decide what are the criteria for successful vaccination. There are three factors to be considered: (i) local reaction to vaccination, (ii) degree of tuberculin sensitivity produced, (iii) response to the diagnostic B.C.G. test.

Local Reaction to Vaccination.

Table V shows the size of the reaction on initial vaccination in 1949 and on revaccination in 1951 of 27 "revertors". These figures suggest that the smaller the local reaction, the greater the risk of reversion to a negative type of tuberculin reaction. Hertzberg has shown that there is a distinct parallelism between local reaction to vaccination and the size of the Mantoux reaction, although it is not absolute. He has also shown that the smallest local reaction can be correlated with the highest rate of failure to produce resistance.

Tuberculin Sensitivity Produced.

Ustvedt states that we do not possess enough information about the value of the degree of tuberculin sensitivity as an index of immunity. It is sometimes extremely difficult to decide whether to class a reaction as positive. The lower limit of the size of a positive reaction is put at a figure varying from five to ten millimetres of infiltration. I use five millimetres as a lower limit. It is essential when discussing the tuberculin reaction to state exactly what figure is being used. The degree of infiltration is also important, as it can vary from very slight, perceptible only on palpation, to an obvious raised area.

Another factor which should be considered is whether the standard for a positive result should be the same for different strengths of tuberculin. There has also been much investigation to decide what strength of tuberculin should be used for the pre-vaccination and post-vaccination tuberculin tests. The standard dose generally used at present is 10 units of old tuberculin. The Second Annual Report of the International Tuberculosis Campaign states that a weak vaccine or a potent vaccine, which has been stored for a long time, will produce some reaction with 100 units of old tuberculin, and even with 10 units if testing is carried out six to ten weeks after vaccination. The evidence in this report suggests that only persons reacting to a small dose of tuberculin have sufficient resistance against virulent infection.

It is possible that five units instead of ten units may be a more suitable dose, as it has been shown that people not reacting to five units can be vaccinated without the occurrence of inconveniently large reactions.

The technique of tuberculin testing is also important. A syringe which leaks will not inject 0·1 millilitre. It is worth noting that 0·1 millilitre given intradermally produces a weal eight to ten millimetres in diameter.

Diagnostic B.C.G. Test.

It has been maintained by some that injection of B.C.G. vaccine itself gives more reliable information than the tuberculin test.

Using a 1 in 200 dilution of B.C.G. vaccine, I injected 0·1 millilitre, intradermally on the inner aspect of the

TABLE IV.

Results of Mantoux Testing Two Months, and One and Two Years After Vaccination.

Year.	Number Vaccinated.	Number Tested After Vaccination.	Number of Positive Reactors.	Results One Year After Vaccination.		Results Two Years After Vaccination.	
				Number Tested.	Number of Positive Reactors.	Number Tested.	Number of Positive Reactors.
1949	266	263	257 (98%)	207	200 (97%)	194	165 (85%)
1950	138	84	83 (99%)	115	107 (93%)		

forearm, first on myself, three nursing sisters and a clerk at the chest clinic, and also on a woman, aged twenty-nine years, wife of a patient, and a woman, aged forty years, whose husband had died recently of renal tuberculosis and who had had a discharging sinus from a tuberculous hip, although there was no pulmonary lesion. These last two have failed to react to 10 units of old tuberculin for some years. Two of the nurses, the clerk and I reacted with a papule four millimetres in diameter in seventy-two hours, and in myself a small pustule developed in approximately a week. The third nurse, whose tuberculin response had been negative for many years, showed no reaction; the

TABLE V.

Reaction to Vaccination of the 27 "Revertors" of 1949.

Time of Testing.	Papule 0·5 Millimetre.	Papule Over 5·0 Millimetres.	Pustule.	No Reaction.	Not Checked.
After vaccination in 1949 ..	17	6	3	—	1
After revaccination in 1951	8	2	14	—	3

woman aged twenty-nine years showed a very severe reaction, an area of infiltration 10 millimetres in diameter being produced in forty-eight hours with much œdema of the whole forearm. No ulceration occurred. The woman aged forty years showed no reaction. At her request she was vaccinated. A very small papule five millimetres in diameter was produced. Two months later the reaction to 10 units of old tuberculin was positive.

The test was then tried on 22 subjects who had been vaccinated and who had yielded doubtful reactions to 10 units of old tuberculin. Of these, 10 showed no reaction and were revaccinated, one reacted with a pustule on the original vaccination, and seven did so on revaccination. There was no evidence of Koch phenomena on revaccination. Three, including twin sisters, were included in the 10. These three were not revaccinated, as initial vaccination and a second vaccination two months later had failed to produce any reaction, either to the vaccination or to the tuberculin test. Nine reacted with what was considered to be a Koch phenomenon, two with areas of infiltration 10 millimetres in diameter and one a pustule 10 millimetres in diameter. These last three were not revaccinated. Five of the remaining six reported for revaccination, and in two there was evidence of a Koch phenomenon, but in none was any inconveniently large reaction produced.

Summary.

1. Analysis of the reaction to vaccination shows that there is considerable variation from batch to batch, even with the same vaccinator.

2. Results vary with different vaccinators.

3. There is a considerable drop in the percentage of positive reactors on retesting after two years.

4. No evidence could be produced to show that there are a lower number of successful vaccinations in the summer.

5. Possible criteria for successful vaccination are: (i) local reaction to vaccination, (ii) the degree of tuberculin sensitivity, and (iii) the response to the diagnostic B.C.G. test.

Conclusions.

1. The fall in the percentage of positive reactors on retesting after two years indicates the need for continued strict control of the administration of the vaccine and for adequate checking of those vaccinated. Such control will always be necessary.

2. The technique of administration is of extreme importance. The State directors of tuberculosis should see that those members of their staff performing vaccination have a satisfactory technique. If the tuberculin testing is done by the nursing staff, attention should also be given to their technique. As much care should be given to the performance of the pre-vaccination and post-vaccination tuberculin tests as to the actual vaccination. Nurses quickly learn a satisfactory technique, but generally the medical staff do not give enough time to teaching them. Far too frequently it is left to the senior sister to instruct new members of the nursing staff.

3. The keeping qualities of the vaccine in the summer require investigation.

4. The local reaction to B.C.G. vaccine, the tuberculin sensitivity, and the diagnostic B.C.G. test can be used to determine whether vaccination has been successful, and in doubtful cases use should be made of all three. Standards should be exacting. It would be better to risk producing an occasional inconvenient local reaction by revaccination than to pass an inadequate vaccination, particularly when exposure to infection may be high, as in the case of nurses.

5. I consider that the greatest danger of B.C.G. vaccination is the production of a false sense of security in those vaccinated, and for this reason in a community where the standard of living is high, the public health services are good, and there is a reasonable, if not an ideal, number of beds for the isolation and treatment of infective patients, it would be better not to vaccinate than to vaccinate inefficiently. Finally, I would say that this paper has been written, not as a criticism of B.C.G. vaccination, but to emphasize that there are still many problems associated with its administration.

Acknowledgements.

I should like to thank Dr. J. Wilson, of the State Agricultural Department, Launceston, for assistance in the presentation of the figures, and Dr. J. Tremayne, Director of Tuberculosis, Tasmania, for permission to publish the paper.

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"Conference on European B.C.G. Programmes", pages 194, 199, 201.

"Second Annual Report of the International Tuberculosis Campaign", pages 106, 123.

Addendum.

I have discussed certain aspects of the above results with Dr. E. North, of the Commonwealth Serum Laboratories, and I should like to thank him for his advice. The following observations which he made may be of interest. He regards 84% of positive reactors to 1 in 1000 old tuberculin after two years as moderately satisfactory.

EXAMINATION OF RAW MILK FOR COXIELLA BURNETI IN THE GREATER BRISBANE AREA OF QUEENSLAND.

By P. E. LEE,

Queensland Institute of Medical Research, Brisbane.

"Q" FEVER was discovered in Brisbane before the last war (Derrick, 1937). During the period of the investigation (1935 to 1942) there were only two out of 129 cases of infection of Brisbane residents not explained by association with meatworks or the laboratory (Derrick, 1944). These two cases drew attention to an area beside Kedron Brook. One of the patients was engaged on relief work and lived in Toombul, just north of the brook, and the other worked in the north-eastern part of Clayfield, just south of it. At that time, the area included much open country, on which dairy cattle grazed.

More recently "Q" fever has been shown to be highly endemic in Southern California, and epidemiological data indicated that dairies and dairy cows were sources of human infection in that area. Huebner *et alii* (1948) showed that large numbers of dairy cows were infected with the disease, and were shedding rickettsiæ in their milk. Recovery of *Coxiella burneti* from raw milk by these workers has considerable significance, in that it could provide a method for mapping out areas in which infection is present. It is on this finding that the present work is based.

Methods.

Each sample of milk obtained was inoculated into two guinea-pigs on arrival at the laboratory, one to three millilitres subcutaneously into one, and three to five millilitres intraperitoneally into the other. Spleens of febrile guinea-pigs, and of the majority of those subcutaneously inoculated, were ground with sterile sand, made up with sterile physiological saline, and inoculated intraperitoneally into new guinea-pigs. The material from the febrile guinea-pigs was passaged as soon as possible after the elevated temperatures were noticed, and that from the others at ten to fourteen days after inoculation. All surviving guinea-pigs were killed at twenty-seven to forty-one days after inoculation, and serum was prepared from the heart blood. These sera were sent to the Government Microbiological Laboratory, where they were tested by agglutination for "Q" fever antibodies by Mr. H. E. Brown.

FIGURE I.

The interrupted line marks the boundary of the City of Brisbane; "+" denotes localities from which test samples were obtained; a *plus* sign within a circle marks the suspect area near Kedron Brook associated with two patients; a circle indicates residences, outside Brisbane, of dairy farmers who had "Q" fever in 1938, 1939 and 1944.

Nature of Samples.

Approximately 88% of the Brisbane milk supply is prepared for public consumption by pasteurization at city milk factories, which act as depots for various dairies in and around Brisbane. The remaining 12% is sold directly to the public, without pasteurization, through milkmen who act as intermediaries between farmer and consumer. These milkmen are called "warm milk vendors".

Samples from individual cows were not tested. Instead, specimens consisted of "pooled" milk—that is, a mixture of milk obtained from some or all cows in a herd. Sixty samples of pooled raw milk were tested from dairies within the boundaries of the City of Brisbane or just outside it. The localities from which these were obtained are shown in the map (Figure I), and include an area adjacent to that in which the two infections noted above were found. Twenty-seven further samples of milk were tested, which were supplied to Brisbane from various localities more distant from the city. Of these 87 samples, 52 were obtained from warm milk vendors, and 35 from city milk factories.

Results.

Some of the guinea-pigs inoculated with the milk samples developed fever, but in no case could this be attributed to "Q" fever. No strain of *Coxiella burneti* was obtained by passage from febrile or afebrile guinea-pigs, and no agglutinating antibodies to this organism were detected in their sera.

However, brucellosis was detected by agglutination with *Brucella abortus* in guinea-pigs inoculated with ten samples of milk.

Discussion.

The City of Brisbane extends from six to fifteen miles out from the General Post Office, and occupies an area of 375 square miles. There are about 300 dairy farms in and just around it, so that the 60 herds tested form a fair sample. They were well distributed geographically over the area.

Animal inoculation to obtain evidence of rickettsial infection has been an accepted method for a long time. The original recognition of "Q" fever depended on the results of the inoculation of human blood and urine into guinea-pigs, and Huebner *et alii* used guinea-pig inoculation of milk extensively in their investigations in Southern California. It is thought, therefore, that the results obtained in the present survey should be valid.

Conclusion.

1. The investigations, so far as they have gone, have not disclosed an endemic focus of "Q" fever within Greater Brisbane, although previous work has shown that there are endemic areas in the countryside north and south of Brisbane.

2. It seems unlikely that the raw milk supply is a significant source of human infection with "Q" fever in Brisbane, which is in accord with Derrick's observations on the occupational incidence of the disease.

Acknowledgements.

The author wishes to express her appreciation to Dr. E. H. Derrick, who suggested, guided and contributed greatly to the planning of the work. She is also indebted to Mr. H. E. Brown and Mr. C. M. Cato, of the State Health Department, and to the Brisbane milk vendors and managers of the various milk factories for their helpful cooperation.

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Reports of Cases.

THE SUCCESSFUL TREATMENT OF SEVERE ACQUIRED HÆMOLYTIC ANÆMIA WITH ADRENOCORTICOTROPIC HORMONE (ACTH).

By ERIC G. SAINT[1] and HILDA J. GARDNER,[2]
Melbourne.

SEVERAL reports on the value of adrenocorticotropic hormone (ACTH) in the treatment of acquired hæmolytic anæmia have appeared in recent literature (for example, Dameshek, Rosenthal and Schwarz, 1951; Gardner, McElfresh, Harris and Diamond, 1951; Davidson *et alii*, 1951; Best, Limiarzi and Poucher, 1951). The presence of abnormal circulating hæmagglutinins in Lederer's anæmia was first described by Widal (1908), but little notice was taken of these auto-antibodies until the Coombs test (Coombs, Race and Mourant, 1945) was applied to cases of acquired hæmolytic anæmia and found to give a positive result in a high proportion. Incomplete iso-antibodies may arise cryptogenically or in the course of some generalized disease of the reticulo-endothelial system, such as Hodgkin's disease, leuchæmia or sarcoidosis. Morton and Pickles (1947) have shown that modification of erythrocytes by trypsin renders them suitable for the detection of incomplete agglutinins, and Rosenthal, Dameshek and Burkhardt (1951) have elaborated this technique to follow serial changes in incomplete hæmagglutinin titre during therapy with ACTH or cortisone.

This paper records observations on a patient with an acute hæmolytic crisis treated with ACTH, on whom serial titrations of auto-agglutinins were made before, during and after treatment.

Clinical Record.

Mrs. J.G., aged thirty-seven years, a housewife, was admitted to the Royal Melbourne Hospital on October 25, 1951, with a history of increasing weakness, dyspnœa and lassitude over the previous nine months. Friends had noticed that she had become slightly jaundiced. There was no family history of icterus. She had been married five years but was childless.

On examination she was obviously anæmic and moderately jaundiced. There was no significant enlargement of the regional lymph nodes. The chest was free from abnormality. The liver was not enlarged, but the spleen was palpated two centimetres below the left costal margin.

Blood and other examinations confirmed the presence of a normocytic anæmia due to active hæmolysis. The hæmoglobin value was 8·4 grammes *per centum*, the red blood cells numbered 2,500,000 per cubic millimetre, and the leucocytes numbered 4000 per cubic millimetre. The proportion of reticulocytes was 6%, the packed cell volume was 25%, the mean corpuscular volume was 100 cubic μ, the mean corpuscular hæmoglobin was 32 micromicrogrammes, and the mean corpuscular hæmoglobin concentration was 32%. The platelets numbered 396,000 per cubic millimetre.

A biopsy from the iliac crest showed considerable hyperplasia of the bone marrow with a high proportion of normoblasts.

The twenty-four hour excretion of stercobilinogen was 200 milligrammes *per centum*. The osmotic fragility of the patient's red cells was within normal limits.

The patient was of blood group A (II), Rh-positive. The Wassermann test failed to produce a reaction and Coombs's test produced a positive reaction. The blood sedimentation rate by the Westergren method was 100 millimetres in one hour.

A splenic biopsy showed that the majority of the cells in the smear were of the lymphocytic-monocytic series; but there was nothing to suggest a histological diagnosis of reticulosis or leuchæmia.

A number of biochemical tests were carried out. The serum bilirubin content was 12 units; the cephalin flocculation test produced a "+++" result; the total serum proteins were 6·7 grammes *per centum* (γ globulin, 1·28 grammes); the serum alkaline phosphatase content was three King-Armstrong units.

A liver biopsy showed no abnormal histological features.

The outstanding features of the patient's progress are shown in Figure I. She was considered to have acquired

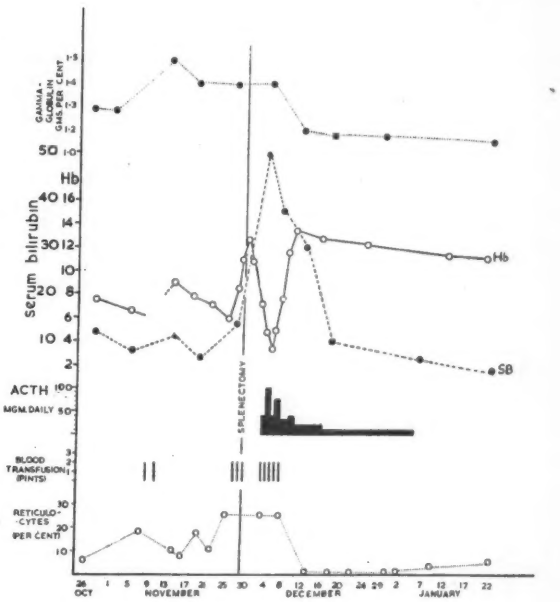

FIGURE I.

idiopathic hæmolytic anæmia, and initially a period of observation was instituted. She had a low-grade fever and the level of hæmoglobin fell significantly from 7·8 to 6·6 grammes *per centum*. After a small blood transfusion the rate of hæmolysis increased (Figure I), and it was decided, in consultation with Mr. Julian Orm Smith, that splenectomy should be performed. As the hæmoglobin value had fallen to 6·0 grammes per 100 millilitres, six pints of compatible blood were transfused over forty-eight hours, and on November 29 an enlarged spleen with two small splenicules was removed.

Immediately after operation there was clinical evidence of a very severe hæmolytic reaction. The patient became restless, distressed and very dyspnœic. The hæmoglobin value fell to 6·0 grammes per 100 millilitres and failed to rise despite further blood transfusion. The proportion of reticulocytes in the blood rose to 26%, and the development of a leucocytosis of 100,000 per cubic millimetre with many nucleated red cells indicated a profound reaction of the bone marrow. Four days after splenectomy the patient was critically ill and her life hung in the balance.

Treatment with ACTH was now begun (December 3). For reasons of economy 20 milligrammes were given intravenously, dissolved in 5% glucose solution, over a period of twenty-four hours. On alternate days 100 milligrammes were given in divided doses by the usual intramuscular

[1] Work done with the aid of a grant from the National Health and Medical Research Council. (From the Clinical Research Unit of the Walter and Eliza Hall Institute of Medical Research and the Royal Melbourne Hospital.)

[2] From the Clinical Pathology Department of the Royal Melbourne Hospital.

route. In view of the possible threat of liver failure, intravenous therapy with 10% glucose solution and protein hydrolysate—"Parenamine" (Stearns)—was maintained. One million units of penicillin were administered prophylactically each day. She received two pints of compatible blood daily for five days.

After two days of ACTH therapy she seemed slightly better, and after four days the improvement in her clinical condition was dramatic. The serum bilirubin level had fallen precipitously, and the hæmoglobin value rose to 15·9 grammes per 100 millilitres and remained there despite discontinuance of blood transfusion. The proportion of reticulocytes fell, and nucleated red cells disappeared from the blood film.

ACTH therapy was continued at a lower dosage until January 4, 1952, for a total period of four weeks. At first 20 milligrammes were given intravenously each day, then 10 milligrammes.

Electrocardiographic evidence of potassium deficiency (prolonged *Q-T'* interval and flat *T'* waves) developed on the seventh day of ACTH administration. She also became œdematous, but had massive diuresis following rigid restriction of sodium intake. On the eighth day of ACTH treatment signs of consolidation were noted at the base of the left lung; but it was difficult to say whether the associated signs of radiological opacity were due to segmental collapse or to a silent pulmonary infarct.

At the end of her course of ACTH treatment she felt very well and was only a trifle jaundiced. The hæmoglobin level had flattened out at a sustained level of 12 grammes per 100 millilitres. After withdrawal of ACTH there was no clinical or hæmatological evidence of relapse, and the patient was discharged to her own home on January 29.

A month later she was examined again in the outpatient department. She was doing her own housework and felt extraordinarily well. She was not jaundiced and the hæmoglobin value was 12·0 grammes per 100 millilitres.

Auto-antibody Studies.

Tests on the patient's serum were carried out according to the technique of Rosenthal, Dameshek and Burkhardt (1951).

The patient's serum diluted in saline and in 20% albumin solution was set up against her own untreated and trypsinized cells and against homologous red cells at temperatures of 3° C., 22° C. and 37° C. Tests were carried out as follows: (i) eight days after her admission to hospital; (ii) seventeen days after the onset of ACTH therapy; (iii) fourteen days after the cessation of ACTH therapy; and (iv) during remission two months after cessation of ACTH therapy.

The results are shown in Table I. During the preliminary period of observation (November 8, 1951) the patient's serum is seen to contain non-specific agglutinins, probably of the "incomplete" type, in so far as they were more readily demonstrable in the presence of albumin, at least at 22° C. Test cells treated with trypsin and exposed to saline dilutions of serum reacted with agglutinins to slightly higher titre than with albumin. Furthermore, the agglutinability of trypsinized red cells was most strikingly demonstrated at 3° C.; this confirmed the reports of other observers (for example, Bouroncle, Dodd and Wright, 1950).

During treatment with ACTH there was a remarkable drop in the titre of auto-agglutinins and iso-agglutinins. The highest titres were demonstrated with trypsinized test cells.

After ACTH therapy had been stopped agglutinin titres rose. However, the pattern was different from that which was described before treatment commenced, for agglutination of test cells suspended in saline was not now demonstrated—only trypsinized cells were agglutinated. Two months after ACTH treatment had ceased a further fall in titre was recorded.

Discussion.

Investigations performed on this patient showed that her hæmolytic anæmia was truly of idiopathic type, for no source of chronic infection was discovered and the histological appearances of material obtained at liver, spleen and marrow biopsy ruled out the possibility of either a reticulosis or leuchæmia. Reports of hæmolytic anæmia in association with these latter conditions, and also with sarcoidosis (Crane and Zetlin, 1945), chronic liver disease (Watson, 1939) and a variety of other toxic and neoplastic states (Wintrobe, 1946) indicate the necessity of a high level of diagnostic accuracy.

TABLE I.

Serial Changes of Titre of Auto-agglutinins and Iso-agglutinins in a Case of Acquired Hæmolytic Anæmia Treated with ACTH.

Temperature of Test. (Degrees Centigrade.)	Treatment of Cells Tested.	Titre. Before Therapy 8.11.51.	During Therapy 20.12.51.	After Therapy 22.1.52 (2 Weeks).	After Therapy 28.2.52 (8 Weeks).
37	*Auto-agglutinins*				
	With saline..	32	0	0	0
	With albumin	32	NP[1]	NP	NP
	With trypsin	128	4	128	32
	Iso-agglutinins				
	With saline..	8	0	0	0
	With albumin	8	NP	NP	NP
	With trypsin	16	16	128	32
22	*Auto-agglutinins*				
	With saline..	64	2	0	0
	With albumin	128	NP	NP	NP
	With trypsin	32	16	128	32
	Iso-agglutinins				
	With saline..	8	2	0	0
	With albumin	2	NP	NP	NP
	With trypsin	128	8	128	32
3	*Auto-agglutinins*				
	With saline..	512	2	0	0
	With albumin	256	NP	NP	NP
	With trypsin	256	64	32	64
	Iso-agglutinins				
	With saline..	512	0	0	0
	With albumin	256	NP	NP	NP
	With trypsin	512	16	32	32

[1] NP = test not performed.

Acquired hæmolytic anæmia runs a variable course, often with long periods of spontaneous remission. In consequence it is not easy to interpret the efficacy of splenectomy in causing prolonged remissions, although most observers with wide experience are convinced of its value even in so-called "symptomatic" cases. In this case the decision to undertake splenectomy was made only after mature consideration, when it appeared that active hæmolysis was endangering the patient's health.

The amount of blood these patients should be given in hæmolytic crises has been a moot point, for it is thought that massive transfusion only accelerates hæmolysis. However, no surgeon or anæsthetist is happy to allow operation on a severely anæmic patient; this patient was therefore given a generous pre-operative blood transfusion. There is no doubt that this was materially responsible in provoking a severe, nearly fatal hæmolytic reaction.

The administration of ACTH was a timely and life-saving procedure. There is convincing evidence, adduced from the fall in serum bilirubin level and the sustained elevation of the hæmoglobin value, that it was the effect of ACTH rather than the delayed effect of splenectomy which so promptly suppressed hæmolysis.

For reasons of economy ACTH was given on alternate days by the intravenous route, 20 milligrammes being dissolved in one litre of glucose (5%) in distilled water given in a period of twelve hours. Mandel, Singer, Gudmundson, Meister and Modern (1951) have shown that this intra-

venous dose is equivalent to 100 milligrammes given by the intramuscular route.

In-vitro tests showed that ACTH caused a striking suppression of auto-agglutination. Whether ACTH acts by causing physiological alterations in the surface of the red cell or by inhibiting the production of auto-antibodies is not known with certainty. In support of the latter hypothesis in this case was the observation of a significant fall of the level of γ globulin (see Figure I) *pari passu* with ACTH therapy.

It is abundantly clear that ACTH offers great hope in improving the prognosis of acquired idiopathic hæmolytic anæmia. Since the spleen is the site of red cell destruction, splenectomy must still be regarded as the cornerstone of treatment, offering the best hope of prolonged remission. ACTH should be given as a cover to suppress hæmolysis, which may be accelerated by blood transfusion; it should be administered two or three weeks before splenectomy, to allow the patient's hæmoglobin value to be brought up to optimal levels, and should be continued in moderate dosage for a few weeks post-operatively. From the experimental evidence available it seems likely that ACTH should control the hæmolytic relapses which may occur at a later date.

Summary.

A patient suffering from acquired hæmolytic anæmia with a severe hæmolytic reaction following transfusion and splenectomy has been treated with ACTH with dramatically successful results. Even after cessation of ACTH therapy the patient has remained in clinical remission.

In-vitro studies with the patient's serum showed that during treatment with ACTH auto-agglutinin activity was significantly suppressed. At the termination of treatment agglutinin titres rose, but to reduced levels.

It is suggested that ACTH should be given prior to splenectomy, to suppress the hæmolysis which may be accelerated by transfusion and to allow the operation to take place under optimal conditions, and for at least three weeks post-operatively.

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Reviews.

PREVENTIVE MEDICINE AND HYGIENE.

A NEW edition of a well-known text-book makes its appearance after a lapse of sixteen years with the publication of the seventh edition of Rosenau's "Preventive Medicine and Hygiene".[1] For this edition, Professor Rosenau has entrusted the production to Kenneth F. Maxcy, Professor of Epidemiology at the Johns Hopkins University, and a new team of collaborators. The book, roughly the same size as before, opens with Section I, "Prevention of Communicable Diseases", and proceeds to deal with the major infectious diseases. Comparison shows a practically new presentation of the subject matter with only an occasional paragraph retained from the previous edition. Selecting at random amongst the new material included, one notices the modern concepts of erythrogenic toxins and the futility of isolating streptococcal infections only when they happen to give rise to a rash, discussed under "Scarlet Fever and Hæmolytic Streptococcal Infections". The value of mass radiography, tuberculin testing and B.C.G. vaccination is assessed under "Tuberculosis"; the relationship of maternal rubella and congenital defects is discussed under "German Measles"; and revised technique in vaccination is described under "Vaccination and Smallpox". A coloured plate illustrates the reactions to vaccination, and here a criticism may be made that the immune reaction does not persist to the extent illustrated at one week; also that in using the multiple puncture technique one should not inflict as much trauma as that shown.

Instead of the deficiency diseases being included in a chapter set amongst the communicable diseases, they are now included in a separate section on "Nutrition and Deficiency Disease"—a more logical arrangement.

Section III deals with the "Maintenance of Health and Prevention of Disability", including infant and maternal health services, school health services and provision for the physically handicapped child, together with chapters on chronic disease and disability in adults, and senescence. Mental hygiene is also considered in this section. As the author states in his preface, the book here reflects "the increasing attention . . . being given to the prevention of diseases of non-infectious etiology, to the maintenance of health in middle and old age and provision . . . for rehabilitation". Another example of the changed emphasis is seen in the disappearance of the section on disinfection.

The sections in the older edition which dealt with "Food", "Air", "Soil", "Water and Sewage" are replaced by new sections on "Food Sanitation", "Environmental Medicine" and "Sanitary Control of Water Supplies, Sewage and Refuse Disposal". The treatment of these subjects is up to date and succinct, and, although it occupies fewer pages, appears to comprise the essentials from the previous edition in addition to some new information of value.

The table of contents is not as complete as might be wished. Should one desire to look up a disease which occupies many pages of the book and has a complete subsection devoted to it, reference to the index is necessary and the new index occupies less than a third of the space of the old. Of course, these are minor matters, but the resultant extra difficulty of reference may be exasperating at times—and such a book is more often used for reference than merely to be read. However, the new edition is, on the whole, an excellent production and a welcome addition to the literature of preventive medicine.

SUPPLEMENT TO THE BRITISH PHARMACEUTICAL CODEX.

THE 1952 Supplement to the British Pharmaceutical Codex[2] follows the general plan of the Codex in its division into the customary sections: "General Monographs", "Antisera and Vaccines", "Preparations of Human Blood", "Surgical Ligatures and Sutures", "Surgical Dressings", and "Formulary".

[1] "Rosenau: Preventive Medicine and Hygiene", by Kenneth F. Maxcy, M.D., Dr.P.H.; Seventh Edition; 1951. New York: Appleton-Century-Crofts, Incorporated. 10″ × 7″, pp. 1478, with 49 illustrations and text figures.

[2] "The British Pharmaceutical Codex, 1949. Supplement 1952", published by direction of the Council of the Pharmaceutical Society of Great Britain; 1952. London: The Pharmaceutical Press. 9″ × 5½″, pp. 160. Price: 25s. net.

Many of the new monographs (Part I) relate to substances such as æthinyl œstradiol, chloramphenicol, cetrimide, isoprenaline sulphate, and many others which were adopted by the British Pharmacopœia in the Addendum 1951. In addition to this group, twenty-two new monographs are devoted to drugs which have not as yet acquired pharmacopœial status, but which, on adequate clinical evidence, are deemed worthy.

Among the better known drugs are the following: amidone ("Methadone", "Physeptone"), a rival of morphine as an analgesic; aureomycin, the wide therapeutic spectrum of which is well attested; vitamin B_{12} disguised under the "approved" alias of cyanocobalamin; dexamphetamine, the dextro-rotatory and less active form of the adrenergic amphetamine—popularly used to make a restricted food intake less irksome for the obese; sodium aminosalicylate, a nearly neutral salt of PAS, so effectively linked in therapy with the streptomycins; troxidone ("Trimethadione", "Tridione") for the control of *petit mal* and of pyknoleptic incidents; and gamma benzene hexachloride, a descriptive if somewhat cumbersome name for the potent insecticide and larvicide better known as gammexane.

Two sulphones—relatives of the sulphonamides—are added as rivals to chaulmoogra products for the treatment of leprosy. These are the simple diamino-diphenyl-sulphone-dapsone, appearing both as a tablet and as an oily injection, and a water-soluble, sodium bisulphite homologue, solapsone—better known as sulphetrone.

The muscle relaxant group has many representatives. These include decamethonium iodide (C10)—a relaxant which, unlike tubo-curarine, is not antagonized by anti-cholinesterases of the neostigmine class; gallamine triethiodide ("Flaxedil")—more closely comparable with tubo-curarine, but compatible with thiopentone and administrable coincidently from the same syringe; mephenesin ("Myanesin"), one of the many simple mono-ethers of glycerol, the future of which would appear to be for the symptomatic relief of spastic, hypertonic and hyperkinetic conditions of the *paralysis agitans*, cerebral palsy and choreo-athetosis types. Of ganglionic paralysants, pentamethonium iodide (C5), tetraethylammonium bromide (T.E.A.B.) and solutions of tetraethylammonium chloride have been selected.

Among newly included preparations for local application are aluminium powder which, incorporated with zinc oxide into a paste with liquid paraffin, is applied to the skin as a protective against proteolytic and irritant discharges; oxidized cellulose, an absorbable hæmostatic which forms a coagulum comprising salts of its constituent polyanhydroglycuronic acid with hæmoglobin; and absorbable gelatin sponge, another hæmostatic composed of formaldehyde gelatin foam.

With the apparently limitless multiplication of chemically complex synthetic drugs, nomenclature is becoming a nightmare—no less, we feel, for those charged with the invention of approved names than for the overburdened practitioner who has to memorize them for prescribing. Many, such as cortisone, dapsone and methoin, come easily to mind and fall trippingly from the tongue, but names like diaethylcarbamazine (for hetrazan) and cyanocobalamin (for B_{12}) are a direct invitation for manufacturers to coin something simple and catchy in the way of a trade name.

A very useful feature of the British Pharmaceutical Codex Monographs is the informal section in each devoted to action and uses. In these sections not only are the actions and uses concisely portrayed, but each is leavened with practical observations relating to the pharmacy, dosage, mode of administration and often treatment of the overdosage of the drug under review. Where the British Pharmacopœia (with increasing frequency) evades the responsibility of quoting specific dosage by stating, "the dose is determined by the physician in accordance with the needs of the patient", the practical, if unofficial, posological guidance of the British Pharmaceutical Codex is the more welcome.

The changes in Parts II, III and IV are for the most part amendments that bring the special monographs in those sections into line with the British Pharmacopœia 1948 and its Addendum. Many new surgical dressings are quoted in Part V and acceptable standards formulated.

The use of rayon as a partial replacement for cotton is sanctioned and a monograph is devoted to a rayon rubber elastic bandage. To retain Latinized pseudonyms for these products—for example, *Carbasus Absorbens in Tœnia* for absorbent ribbon gauze—is surely a relic of Victorian pomposity and completely out of alignment with modern views and work-a-day custom.

The formulary section (Part VI) contains formulæ for injections, nebulæ and tablets incorporating several of the new drugs in Part I. Many older formulæ have been modified for pharmaceutical or therapeutic reasons. Borax and formaldehyde solution, as a surgical instrument preservative solution, has been replaced by a solution of sodium benzoate and chlorocresol.

PHOTOELECTRIC SPECTROPHOTOMETRY.

IN this modern age when science and medicine have gained the interest and respect of society, the importance of spreading facts in print cannot be denied. The difficulties and expense of publication have usually acted as serious deterrents to authors, but after studying this book one is led to conclude that the golden age has really arrived, at least in the United States of America. It is embarrassing to find oneself mentally asking, whilst reading, why this book was published.[1]

As an interpretation of the origin and development of the book and without any intention of belittling either the subject or the author's profession, it would appear that a clinical practitioner has suddenly seen the light, of monochromatic variety, and after learning of its value in spectrophotometry, has become imbued with an intense desire to spread the good news.

There is little to criticize regarding the accuracy of the contents, but there is very little justification for the laborious details. Who should benefit from the book? Perhaps the clinical worker; but if that is so, then the development of the subject matter is open to serious criticism. Many pages are wasted in giving detailed solutions of simple algebraic equations, yet no useful advice is offered about instruments for the measuring of light absorption. Whole pages are taken up in describing a few experimental conditions for adjacent absorption curves *et cetera*. In fact, the latter part of the book is devoted to the reproduction of absorption curves of various hæm compounds, and to nomograms. Whoever wishes to work with the blood pigments will need to prepare his own graphs, so there is no real excuse for presenting them on such a grandiose scale.

The book is well printed on expensive glossy paper, thus creating a superficial impression of quality. Although many workers may find it useful, the superior finish has made the price greatly exceed its true value.

GYNÆCOLOGY.

"TEXTBOOK OF GYNÆCOLOGY", by Wilfred Shaw, has always commanded respect by virtue of the status of the author in his branch of the profession, and this sixth edition brings the work right up to date.[2]

While there are no drastic changes from the fifth edition, this sixth edition is completely revised where modern trends have indicated the need for revision. The book is well set up and printed on good paper with easy-to-read type, and the photographs are excellently reproduced, particularly some of those which are in colour.

The opening chapters deal with the anatomy and physiology of the female generative organs, including their embryological development. Perhaps this section of the book is more suitable for medical students, for whom it has been simplified, but in the later chapters much valuable clinical material is to be found by the practising gynæcologist. Changes in the endometrium during the menstrual cycle are well illustrated by photomicrographs.

A good feature of the book is that when the author discusses diagnosis and treatment of any disease he deals with the appropriate operation, if an operation is necessary, in requisite detail in the pages immediately following. This is a far sounder teaching method than the adoption of a separate section of operative gynæcology.

[1] "The Quantitation of Mixtures of Hemoglobin Derivatives by Photoelectric Spectrophotometry", by Francis T. Hunter, A.M., M.D.; 1951. Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 9¼" × 6¼", pp. 246, with 6 figures and 42 charts. Price: 63s.

[2] "Textbook of Gynæcology", by Wilfred Shaw, M.A., M.D. (Cantab.), F.R.C.S. (England), F.R.C.O.G.; Sixth Edition: 1952. London: J. and A. Churchill, Limited. 9" × 6", pp. 680, with four plates in colour and 304 text figures. Price: 27s. 6d.

Vaginal examination is described in detail, including illustrations, in the left lateral position. In this country we feel that the lithotomy position is much more informative and not obnoxious to the patient when she is properly draped.

The chapter on diseases of the urinary system contains much useful clinical material and is a résumé of current medical practice—howbeit of a rather elementary nature. No mention is made of the urea concentration test or blood urea estimation, and X-ray diagnosis—especially noticeable in regard to pyelitis—is completely neglected.

The section of the book dealing with sterility is very sketchy and the photographs of salpingograms are very poor. Were it not for the captions it would be difficult to say which tubes were occluded and which showed intraperitoneal spill. In our experience, and with the use of a suitable cannula, it is not necessary to pass Hegar's dilators prior to the preparation of a salpingogram, and resultant faintness and discomfort are reduced to a minimum. In the treatment of sterility it is surely dangerous to teach students that a small fœtal uterus means a hopeless prognosis and needs no further investigation. How often does such a patient go out and make one look foolish by becoming pregnant!

A very unusual feature of this book is its frank discussion of normal coitus—and a section on contraception.

It is stated that the Grafenberg ring (with illustration) is used extensively at the present day and is fairly reliable, but the introduction of a foreign body into the uterus, with resultant low-grade endometritis, would be condemned by the majority of Australian gynæcologists.

In the treatment of habitual abortion it is surprising that no mention is made of large doses of stilbœstrol. This seems to be the most successful therapy to date. However, the whole section of abortion is very well summarized and presents a complete picture.

Perhaps an operator who perforates the uterus during curettage of a septic abortion might be rather drastic in carrying out an immediate hysterectomy as advised. Conservative treatment seems to have its place nowadays—thanks to the antibiotics.

That part of the book dealing with the disorders of menstruation is particularly well written and will be of great value to anyone practising clinical gynæcology. No extravagant claims are made, but the general résumé of treatment is both sound and thorough. Radiological treatment in gynæcology is modernized in a chapter dealing with X rays and radium and their clinical indications. With such an authority as Wilfred Shaw, it is only natural that the section of the book dealing with ovarian tumours should be one of its highlights. Nor is the reader disappointed.

This volume can be confidently recommended—more especially to final year students and to young resident medical officers in the gynæcological wards.

EAR, NOSE AND THROAT DISEASES.

IT has been estimated that in general practice diseases of the ear, nose and throat outnumber those of any other region. Some of the details of diagnosis and treatment require special equipment and training and constant practice, and so do not concern general practitioners. Any standard text-book *plus* out-patient experience would provide them. Dr. McKenzie, however, in his short book aims to produce ready-to-hand methods and succinct practical advice.[1]

The subject matter gives a balanced perspective of the common ear, nose and throat diseases, following modern, widely accepted principles. The ear, nose and throat surgeon might not agree with minor points, but he must approve its general soundness. The text is profusely interspersed with illustrative case histories. The only two pictures, while appealing to the eye, have little to offer from a medical point of view that could not have been better described in words. There is a fairly long chapter on deafness and hearing aids which is practical and informative. The author apologizes for his description of audiometry, but feels that it helps. He himself includes a copy of the "audiograph" in his report to a doctor.

In his short chapter on prescriptions—and elsewhere for that matter—Dr. McKenzie adopts an iconoclastic attitude towards many accepted methods, but consoles himself and possibly others with the placebo that "as they do no harm, it is scarcely worth while to persuade anyone not to use them".

For a small outlay this book will bring a rich return in common-sense guidance.

TUMOURS OF THE SKIN.

THE publication of a second edition of "Tumors of the Skin" is especially welcome in this country.[1] Although the subject matter is covered somewhat in the large dermatological text-books, this is the most comprehensive work in English that we can recall. One can buy an expensive book with abundant pictures, but with a disappointing lack of technical detail as to treatment. Here at last is an up-to-date volume that should satisfy any expert from whatever angle it is viewed. This does not mean that one could agree with all the methods of treatment, but the point is that the reader has a clear understanding of what the authors practise down to the finest detail.

The sections dealing with the pre-cancerous and cancerous conditions of the skin will be studied with the greatest interest owing to their high incidence in Australia.

Space will allow only for a brief comment on one or two subjects.

In *kraurosis vulvæ*, vulvectomy is advocated rather than the doubtful procedure of radiotherapy. With reference to this and allied diseases a brief mention only is made of *lichen sclerosus et atrophicus*—a condition of increasing diagnostic importance.

In the experience of the authors "Bowen's disease" responds to radiation and we agree.

For neoplasms such as rodent ulcers and small epitheliomata the writers prefer to work with relatively low voltages such as 100 kilowatts when using X-ray therapy. They frequently mention an initial dose of 3000*r*, followed in a week by 2000*r*. Almost in the "next breath", if not in the next paragraph, they advise divided doses such as 500*r* or 1000*r* every other day up to 7000*r* as required. Most Australian dermatologists and radiotherapists prefer divided dosage in preference to a massive application in order to get good cosmetic results and lessen the risk of necrosis.

For the novice and the expert there is a particularly good detailed account of plastic surgery.

For the actual treatment of a neoplasm there is a practical summary of the advantages and disadvantages of the therapeutic measures employed. These are in the main radium therapy, X-ray therapy, surgical diathermy, surgical excision and plastic surgery.

A feature of the book is the excellent illustrations whether clinical, histological or therapeutic.

This book is highly recommended to all interested in the treatment of cutaneous neoplasms. The only criticism is the price, but this is not too high for those workers who desire to possess a volume of outstanding merit. The binding, printing, references and index are worthy of the publication. It is to be regretted, however, that no references have been made to two outstanding authorities in this country, Molesworth and Paul.

UNTOWARD REACTIONS OF CORTISONE AND ACTH.

IN the very brief compass of 51 pages Derbes and Weiss have set out the less desirable effects of a new and widely used method of therapy. "Untoward Reactions of Cortisone and ACTH"[2] is published in the American Lecture Series

[1] "Ear, Nose and Throat Diseases: For the General Practitioner", by William McKenzie, M.B., B.Chir. (Cantab.), F.R.C.S. (England); 1952. Edinburgh and London: E. and S. Livingstone, Limited. 7¼" × 5", pp. 144, with two plates and eight text figures. Price: 9s.

[1] "Tumors of the Skin: Benign and Malignant", by Joseph Jordan Eller, B.S., M.D., and William Douglas Eller, M.D.; Second Edition; 1951. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9¾" × 6¾", pp. 698, with 550 illustrations and three plates in colour. Price: £8 1s.

[2] "Untoward Reactions of Cortisone and ACTH", by Vincent J. Derbes, M.D., F.A.C.P., and Thomas E. Weiss, M.D.; 1951. Oxford: Blackwell Scientific Publications. 9" × 6", pp. 55. Price: 8s. 6d.

edited by Roscoe L. Pullen. The material used is drawn from recent contributions to the vast literature on this subject, the work of the authors being in the main to coordinate and systematize rather than to offer any personal opinions or advice.

A short but comprehensive review of the currently held views on the physiology of the glands of internal secretion with special reference to cortisone and ACTH is followed by a series of chapters setting out the "untoward effects". These are described under such headings as "Electrolytes", "Infections", "Gastro-Intestinal Tract" and "Nervous System". The effects of therapeutic and experimental dosages are given, both in human subjects and in experimental animals. This results in a rather confusing combination of experimental physiology and clinical medicine which detracts somewhat from the value of the book. Many of the untoward reactions to these agents may be predicted when the dosage in common use exceeds by so much the physiological amounts secreted by the adrenal cortex and the anterior pituitary lobe. Less easily predicted, and therefore of greater interest, are the effects on the central nervous system and on the body's defences against infections. The experimental evidence of interference with the reactions of immunity, not only to tuberculous but also to virus infections such as poliomyelitis, mumps and influenza, are very well reviewed. The changes in the electroencephalogram and psychiatric complications of a serious nature are reviewed in a way which suggests that real danger exists in many cases when patients with even slight personality defects are treated with cortisone or ACTH.

This book should serve to discourage those who might otherwise undertake too lightly the treatment of patients with new, potent and comparatively untried therapeutic agents. While one cannot gain from the compilation of untoward results a truly philosophical concept of the balance of benefit over dangers, it is well to be reminded that the use of cortisone and ACTH is not without risk to the patient.

MALIGNANT DISEASE AND ITS TREATMENT BY RADIUM.

THOSE interested in malignant disease will note with pleasure the publication of the fourth volume of Sir Stanford Cade's book "Malignant Disease and its Treatment by Radium".[1]

For the second edition there have been added two new chapters on the reticuloses and on the leuchæmias. The other chapters deal with malignant disease of the skin, kidney, testis and prostate, bone sarcoma, sarcoma of soft tissue and intracranial tumours.

As with other volumes, the natural history, sites, ætiology, histopathology and clinical types of the various tumours are discussed, and these sections of the book alone should make it of interest to every clinician in the cancer field. However, the real worth of the book would appear to be in those sections devoted to the choice of the method of treatment, whether surgery or radiotherapy or a combination of the two. X-ray therapy is given its place as well as radium therapy. In malignant melanoma, whenever possible, extensive and radical operation is advocated, but this does not mean that this tumour is always unsuitable for irradiation. Irradiation is often worth while in cases unsuitable for surgery. Clinical histories showing regression of melanomatous lesions after irradiation are given. Simple orchidectomy with irradiation of the abdominal lymph nodes has become the method of choice in malignant testicular tumours (both seminoma and teratoma), and considerable improvement in the results has followed.

An excellent detailed account of osteogenic sarcoma is given, and the position of radiotherapy in its treatment defined. It is considered legitimate, as some cases respond well to radiotherapy, to give it as a preliminary to amputation, particularly in view of the poor results from surgery alone. There is no evidence that this adversely alters the patient's expectation of life. Amputation can be done later, in some cases postponed for a lengthy period. In Ewing's tumour, a highly radiosensitive tumour, irradiation should be the first method of treatment. The advantages and disadvantages of surgery and radiotherapy in osteoclastoma are discussed.

In sarcoma of soft tissue, wide excision should be carried out whenever possible, but it is considered that prognosis is improved by either pre-operative or post-operative irradiation. Lengthy chapters on the reticuloses and the leuchæmias cover all the known methods of treatment, including the latest chemotherapeutic agents and radioisotopes as well as the conventional X-ray therapy. There is much of interest in the last section on intracranial tumours, as it indicates the response of various tumours to irradiation, and includes details of radiation technique.

Analyses of the results of treatment of the various types of lesions are given throughout the book, and many histories of illustrative cases. There are numerous excellent illustrations.

[1] "Malignant Disease and its Treatment by Radium", by Stanford Cade, K.B.E., C.B., F.R.C.S., M.R.C.P., F.F.R. (Hon.), with a foreword by Ernest Rock Carling, F.R.C.P., F.R.C.S., F.F.R.; Volume IV, Second Edition; 1952. Bristol: John Wright and Sons, Limited. 9½" × 6¼", pp. 558, with 785 illustrations, a few in colour. Price: 63s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Oculorotary Muscles", by Richard G. Scobee, B.A., M.D., F.A.C.S.; Second Edition; 1952. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10" × 6½", pp. 512, with 159 illustrations. Price: £5 15s. 6d.

The author's aim has been "the presentation of a relatively simple, logical approach to the diagnosis of dysfunction of the oculorotary muscles".

"The 1951 Year Book of Endocrinology (January, 1951-January, 1952)", edited by Gilbert S. Gordan, M.D., Ph.D.; 1952. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 416, with 102 illustrations. Price: $5.00.

One of the "Practical Medicine Series of Year Books".

"The Medical Clinics of North America"; 1952. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9" × 6", pp. 316, with 37 illustrations. Price: £7 5s. per clinic year in cloth binding and £6 per clinic year in paper binding.

This is a "New York Number" and comprises a symposium of 18 articles on endocrine and metabolic disorders, with a foreword by Louis J. Soffer.

"The 1951 Year Book of Pathology and Clinical Pathology (January-December, 1951)": Pathology, edited by Howard T. Karsner, M.D., LL.D.; Clinical Pathology, edited by Arthur Hawley Sanford, M.A., M.D.; 1952. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 454, with 147 illustrations. Price: $5.50.

One of the "Practical Medicine Series of Year Books".

"Aids to Biology", by R. G. Neill, M.A.; Third Edition; 1952. London: Baillière, Tindall and Cox. 6½" × 4¼", pp. 300, with 21 text figures. Price: 6s.

One of the "Students' Aids Series"; the second edition was published in 1948.

"Dental Practitioners' Formulary, 1952: For Use in the National Health Service". London: The British Medical Association and The Pharmaceutical Press. 7" × 4½", pp. 28. Price: 1s. 6d.

The first edition appeared in 1949; it has now been revised in the light of experience.

"The Human Pelvis", by Carl C. Francis, A.B., M.D.; 1952. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9" × 6", pp. 210, with 61 illustrations, three in colour. Price: 52s. 6d.

Based on a course in applied anatomy designed for surgical resident medical officers and for practising surgeons.

"The Story of the Adaptation Syndrome: Told in the Form of Informal, Illustrated Lectures", by Hans Selye, M.D., Ph.D. (Prague), D.Sc. (McGill), F.R.S. (Canada); 1952. Montreal: Acta, Incorporated. 9" × 6", pp. 226, with about 70 illustrations.

The book is a "brief summary" of the author's work and it is "intentionally quite subjective and personal".

The Medical Journal of Australia

SATURDAY, AUGUST 30, 1952.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE CONCEPT OF NORMAL.

In any discussion on disease the concept of normal is sure to be mentioned, disease being some departure from the normal. Medical treatment thus aims to return the patient to normal or to some condition not too far removed from normal. What then is meant by the term normal? Many definitions have been given, but none of them is wholly satisfactory. Gerda Seidelin, writing from the Institute of Medical Biochemistry, University of Copenhagen, has attempted to clarify the position and has discussed various meanings which have been given to the term normal.[1] Her paper attracts attention by its title and the reader turns to it hopefully for enlightenment. The three definitions which she quotes first are worthy of repetition, particularly the first two, of which we give free translations from the German and French respectively. H. Rautmann wrote in 1921 that the expression normal gave information about frequency of occurrence from which varying conclusions could be drawn. P. Dally wrote in *La presse médicale* in 1937 that a normal man might be provisionally defined by the following formula: he is certain to maintain good physical and moral health, he is destined to enjoy a satisfactorily long life, he is capable of moderate activity and of giving a good social account of himself, he constitutes a good quality reproducer of his kind and in illness he behaves well. In 1938 D. Mainland wrote that in medicine and allied sciences (apart from its use by statisticians to denote a curve form) "normal" had been used to denote (*a*) what occurred "as a rule", that is, most commonly, and (*b*) healthy. He declared that if anything was not normal in the first sense, there was a tendency to think of it as abnormal in the second—"diseased or verging on disease". A fourth definition quoted by Seidelin shows that the authors concerned leave us as much in the dark as we were when we started on the inquiry—Clark W. Heath (1945) defines normal as the balanced harmonious blending of functions that produce good integration. Many kinds of such integrations, he declares, are reflected in widely divergent types of personality and behaviour. Out of the superabundance of definitions Seidelin has drawn up what she calls an arbitrary classification of the ways in which the term normal is applied; she has done this to provide a basis for discussion. She has three groups:

1. A standard in physics, chemistry *et cetera*, for example, "a normal solution".

2. An average value pertaining to a certain group, the term being applied to: (*a*) averages of attributes in "healthy" individuals; (*b*) averages in any group, healthy, unhealthy or arbitrarily chosen for some specific use; (*c*) an ideal figure, such as the average value *per se*.

3. An indication of what is "optimal"—(*a*) an optimum fairly frequently met with; (*b*) the highest obtainable.

Regarding the first group no comment is necessary. When we consider normal as an average of findings in "healthy" individuals we at once meet the question: What do we mean by healthy? There is a tendency to consider that a person is healthy who is fit and adaptable, but fit and adaptable for what? The attributes sought for in the miner, the typist and the scientific worker are very different. Very many investigations have been made on "normal", "healthy" young men without any attempt to decide what is meant by normal, healthy young men. A point which is commonly overlooked by medical workers is that of variability. A feature of all living things is variability; no two individuals are exactly alike, and among individuals there are variations in the value of any function of the body; these variations are in some factors wide, in others quite narrow. Because of the lack of recognition of "normal" variability a number of physical findings, due to clinical and associated methods, have with quite inadequate justification been considered abnormal, in the sense of pathological. The variability arising from errors in the technique of quantitative determinations is not often considered, and errors in such determinations might be quite large even from first-class technicians. Thus a true red cell count of 4,500,000 may be presented as anything from 4,000,000 to 5,000,000 by a careful worker and with even wider limits by less careful observers. Standardization of conditions of the subject before determinations are made is often lacking, particularly, for example, in basal metabolic rate determinations, but also in the estimation of blood pressure and many others. Because of the very wide range of values for the blood pressure one cannot speak of a normal blood pressure. The fallacy of accepting the average of observations of any function on a number of subjects as the normal has been exposed many times, but still persists. The normal curve of distribution is not too often found in biology, and if the distribution found is skew, averages become quite meaningless. A well-known author, using average value as equivalent to normal, writes that "no two pelves are exactly alike, and perfectly normal pelves are rarely seen". The concept of "optimal" has of late years entered the definition of "normal". A Scandinavian writer, P. Bonnevie (quoted by Seidelin), has expressed this very well. "The hygienic norm is an optimum expressed by the quantitative value of an attribute, including the variability, and derived on a basis of prognostic experience regarding the group of individuals on whom observations were made, to the effect that these individuals have a smaller morbidity, a longer life-span

[1] *Acta medica Scandinavica*, 1952, Supplement 266 to Volume CXLII, page 891.

or a better development, functional ability, adaptability *et cetera* than the individuals of other groups in which the quantitative values were different in spite of their having lived under the same circumstances." This, of course, expresses an ideal, but no such data are available about any function or are likely to be for a long time. Even with this definition we are faced with the question: "Adaptable for what?" It has been shown in dogs that the optimal protein intake varies whether we consider rate of growth, muscular activity, longevity, resistance to cancer *et cetera*. What is to be considered as "optimal" when this is linked with "normal"? Is it the best usually met with or the highest attainable? By either of these criteria there is not and there never has been a "normal" individual. Aiming at an ideal normal can give some curious statements. Thus Seidelin refers to C. Daly King, who defines "normal" as "that which functions in accordance with its inherent design, that is, a pattern norm", and further, "the organic design of the human being is the one complete design of its kind within the entire organic kingdom on this planet . . . thus the human design represents the complete and fundamental organic pattern, from which other species depart by one degree or another".

It will be seen that we are by no means clear as to what is meant by "normal". As we must have some basis to determine what is abnormal, it seems we must use the data available, but with more circumspection than many, including ourselves, may have done in the past. In regard to man himself we must consider the whole individual and not a figure for one particular attribute. The functional element must be taken into account, but we must also ask in what circumstances the individual is to function. What the average individual does is not necessarily considered normal. In a paper read in 1939 before the Medical Society of Individual Psychology, London, R. Hargreaves pointed out that we must not equate normality with either mental health or average behaviour. He said that the normal individual had not necessarily a healthy mind. Normality here was a social judgement of acceptability, whereas mental health was a medical judgement which should not be influenced by the surroundings in which the condition was found. Hargreaves's conclusion was that normality was a condition of acceptability, and that just as standards of normality varied in different societies, so they might vary in different individuals. This discussion which has been based almost entirely on Seidelin's paper may not have led us to any definite conclusions, but it should at least make us aware that when we speak of normal persons, findings or events we must realize the insecure basis of many of our remarks.

Current Comment.

POST-OPERATIVE PAIN.

MANY investigators have sought a means of prolonging local anæsthesia in order to control post-operative pain, as well as in the management of conditions causing localized pain or pruritus. Results have been disappointing. A favoured plan has been to use a vegetable oil as solvent in the hope that the anæsthetic agent would be released slowly over a prolonged period; but as long ago as 1947 Michael Kelly,[1] of Melbourne, demonstrated the inefficacy of two representative preparations of this kind. In addition to the uncertainty and frequent failure of the prolonged anæsthetic effect, more serious disadvantages may attend the use of oily preparations of local anæsthetic agents. For example, A. H. Iason and H. E. Shaftel[1] cite the occurrence of abscesses and of fibrotic and necrotic tissue changes, as well as of encapsulation, these complications being attributed to the solvent and not to the anæsthetic agents. It is surprising then, as they comment, that such preparations are still used by some surgeons for anæsthetic purposes. Instead, they recommend "Efocaine", which is a specially balanced solution of normally water-insoluble anæsthetic agents (procaine and butyl amino benzoate in a non-toxic vehicle composed essentially of propylene glycol and water) stated to prolong anæsthesia for over a week. When it is injected into the tissues, the contact with the body fluid causes deposition of the active ingredients and the production of a depot of anæsthetic material. In a series of 100 cases "Efocaine" was injected through a 22-gauge needle at the end of the operation. The injections were made into the subcutaneous and muscular tissues, care being taken not to introduce the material intravenously or intradermally. Up to 15 millilitres of "Efocaine" were made to encircle the incision or were injected in lines to block the nerves on each side of the incision. In other cases the injections were made at specific points to block the nerves supplying the incision. Of 60 patients submitted to abdominal surgery 27 stated after injection that they had no pain or discomfort in the injected surgical site, 19 had local discomfort on the first post-operative day, and 14 had some pain for the first twenty-four to thirty-six hours. All who had discomfort or pain had undergone procedures which involved visceral manipulation, and the possibility of visceral pain could not be discounted. The post-operative drug requirements were greatly reduced after these injections. A group of 29 patients undergoing ano-rectal or vaginal surgery received injections of "Efocaine", and in no case was post-operative pain experienced. Only six of the 29 patients required sedative medication; and in these instances it was given only for restlessness and to induce sleep. None of the 29 patients needed catheterization. In the whole of Iason and Shaftel's series the anæsthetic duration as determined by the skin prick technique averaged twelve days, with a minimum duration of six days and a maximum duration of eighteen days. Post-operative anæsthesia was observed for more than two weeks in patients in the ano-rectal surgical group; sphincter control, however, was present after the third to fourth day. There was no evidence of interference with wound healing, and no local tissue reaction occurred. There were no instances of encapsulation, foreign body reactions, sterile abscesses or tissue sloughs, as had been reported after use of the oily solutions; and no instance of systemic or local toxic effects was observed.

It is to be hoped that others can reproduce these encouraging results.

[1] *The Lancet*, May 24, 1947.

ANOTHER REPORT ON NURSING.

THE "collective views of an international group of experts" on nursing are the subject of one of the "Technical Report Series" of the World Health Organization.[2] The views are interesting. To begin with, it is pointed out that the health needs of the people include food, shelter, a healthy environment, ability to use available resources, and provision of care for the sick. The committee urges the World Health Organization to emphasize the education of women, particularly in child health and family hygiene, and to make every effort to improve the general health of women in countries in which such education is deficient, in order to raise the status of women and to enable them to make a full contribution to the health needs of their people. Nursing is part of the health service and it will advance as the need for it is recognized and as economic and other resources support it. Nurses have to satisfy needs in

[1] *The American Journal of Surgery*, April, 1952.

[2] Expert Committee on Nursing: Second Report, World Health Organization, Technical Report Series, Number 49; 1952. Geneva: World Health Organization; Australia: H. A. Goddard Proprietary, Limited, Sydney. 9½" × 6¼", pp. 20. Price: 1s. 3d.

hospitals, health centres, clinics, and the homes of the people. In this country this duty is generally recognized, but it may be useful to refer to the four main aspects of nursing described by the committee and to the four main tasks for nursing that are named. The first task has to do with sociological and psychological aspects of nursing. The nurse "relates her health education to the habits and understanding of the people". She has to satisfy the emotional needs of her patients and to stimulate community movements aimed at the improvement of health. The second task is described as the operative aspect of nursing—the provision of care for the sick, the giving of assistance at childbirth, the rehabilitation of patients, and so on. The third task has to do with education—"all nursing personnel have a moral obligation to the community to teach the prevention of illness". The nurse has educational responsibilities to all with whom she comes into contact, including voluntary workers, auxiliary personnel and other nurses. The fourth task named is far-reaching—it deals with administrative and advisory aspects. "Nursing", we read, "is the conscious practice of human relationships." Then we are told that "It begins with the supervisory attitudes of seniors to juniors and culminates in more onerous and complicated administrative functions, such as the planning and organization of new health services and teaching programmes". It is not clear exactly what is meant by this statement. If the contribution of a nurse to a new health service is to be limited to the part of the service that has to do with nursing, most medical authorities will agree. Beyond this, it is doubtful whether the assistance of nurses would be needed.

Two further important points are made in this report. One is that special care must be taken in the initial selection of nursing students in areas where the educational level of the people is low, and that although schools of nursing should provide training sufficiently comprehensive to fit the qualified student to fulfil all aspects of the nurse's role, curricula must be adjusted according to the general stage of development of the students and the communities from which they have been drawn. The second point is that nurses will make the best possible contribution to the maintenance of health if they are trained to carry out their full duties, if they are properly placed in the health service with full understanding of what their contribution should be and what the aims and objectives of the service are, and if they bring to the task the necessary human understanding. There may appear to be a certain amount of incompatibility about these two statements. If the second point is to be gained the nurse will need to have intelligence a little higher than the average. Discrimination is thus needed in the acceptance of the first of the two points. No doubt the report as a whole is applicable chiefly in countries where the training of nurses has not reached a particularly high level. In some other countries there has been a tendency to open more widely the gates of admission to the nursing profession and to admit trainees whose educational standards are not particularly high. This is a pity, for nursing requires a good deal more than adaptability and skill in the carrying out of certain mechanical duties. The discrimination and the understanding of values required of a nurse are not readily picked up by those of inferior education.

Other aspects of the subject are mentioned in this report. One has to do with the granting of funds from educational sources to ensure for nursing schools an adequate and independent budget. This report is commended to those who are interested in the planning of nursing services.

INTRAARTERIAL BLOOD TRANSFUSION.

SPASMODIC INTEREST has been shown in intraarterial transfusion of blood since last century, but intravenous transfusion has largely held the field as being simpler, more convenient and satisfactory for most purposes. Probably because of growing confidence in resuscitative measures and success in their refinement, more attention has been paid recently to the intraarterial route for effecting blood replacement. It is apparent that for certain purposes it has advantages over the intravenous route when rightly used. Writing in 1948, M. R. Porter, E. K. Sanders and J. S. Lockwood[1] reviewed these advantages, but expressed the opinion that some of the benefits attributed to arterial transfusion might be actually due to the rapid rate at which the blood had been administered. They found the relative importance of the route of transfusion, on the one hand, and the rate of administration, on the other, difficult to evaluate. This difficulty seems to some extent an artificial one, as the route and rate can be regarded as going together. J. H. Gibbon and J. W. Stayman,[2] discussing intraarterial transfusion as part of a consideration of the physiology of cardiac surgery, are quite clear about the matter. They state: "The explanation for the better effects obtained by injecting blood rapidly into an artery is quite obvious. By this means the blood pressure is rapidly restored to normal. This immediately restores the coronary blood flow to normal, since the coronary blood flow is dependent upon the aortic blood pressure. Thus the vicious cycle of depleted blood volume, lowered aortic blood pressure, decreased coronary blood flow and hypoxia of the myocardium is at once reversed. On the other hand, when large amounts of blood are transfused rapidly into a vein of a patient with a severely depleted blood volume and lowered blood pressure, a sudden burden is placed on the right side of the heart and then upon the left side of the heart before any improvement occurs in the coronary blood flow. Under these circumstances it is not surprising that patients in profound shock fail to rally with large intravenous blood transfusions." This does not, of course, diminish the value of the slow intravenous administration of blood, which is in many circumstances more appropriate and has advantages in relation to safety and simplicity.

The indications for use of intraarterial transfusion have been summarized by S. F. Seeley and R. M. Nelson[3] in a review of the literature on the subject. As the most important indication they list rapid restoration of the blood volume as a resuscitative measure against the sequelæ of hæmorrhage, such as shock, the agonal state and "clinical death". They point out that with the use of pressure massive quantities of blood can be infused very rapidly, with the added advantage of immediate perfusion of the renal, coronary and medullary arteries. This, it is suggested, represents the most effective device by which one can speedily overcome the disparity between the circulating blood volume on the one hand and the greater capacity of the vascular tree on the other—a state which exists in incipient or established circulatory collapse and must be rapidly overcome if one hopes to prevent the development of oliguria and the vicious circle instituted by hypoxia and further tissue damage. Other indications are the rapid pre-operative preparation of patients in a state of shock, the control of hypotension during cranial surgery, the prophylactic control of blood pressure in cardiac or abdominal surgery, the location of an uncontrolled bleeding artery and the combating of the shock of coronary occlusion. The use of intraarterial blood transfusion in accordance with most of these indications receives enthusiastic support from D. L. C. Bingham,[4] who has had experience of its use in over 100 cases. Rather cautiously he states that this experience "suggests" that intraarterial transfusion is far superior to intravenous transfusion both in the treatment of established shock and as a preventive measure in operations in which serious shock is expected. Personally, however, his experience seems to have had a convincing rather than a suggestive effect, as he goes on to recommend that apparatus for intraarterial transfusion should be immediately available in the operating rooms and casualty departments of all hospitals, in advanced hospitals and field ambulances on active service, and in blood banks and treatment centres of civil defence organizations. Those who wish to follow the matter further will be interested in Bingham's account of the method of administration with apparatus that he describes as "the simplest and most foolproof that we have been able to devise with materials available in almost any hospital".

[1] *Annals of Surgery*, October, 1948.
[2] *The Surgical Clinics of North America*, December, 1949.
[3] *Surgery, Gynecology and Obstetrics*, March, 1952.
[4] *The Lancet*, July 26, 1952.

Abstracts from Medical Literature.

THERAPEUTICS.

Pneumonia and Antibiotics.

H. F. FLIPPIN *et alii* (*The Journal of the American Medical Association*, November 3, 1951) describe the use of aureomycin, chloramphenicol and penicillin in the treatment of bacterial pneumonia. Ninety-three patients were treated with aureomycin, chloramphenicol and intramuscularly and orally administered penicillin. Aureomycin was given in doses of 0·5 gramme by mouth, followed by 0·25 gramme every six hours, the average total dose being 8·0 grammes. The dosage of chloramphenicol was 1·0 gramme *statim*, followed by 0·5 gramme every six hours, with an average total dose of 14·2 grammes. Oral penicillin therapy was given in doses of 500,000 units, followed by 250,000 units every twelve hours, with a total dose of 3,000,000 units. The intramuscular dosage of penicillin was 300,000 units of procaine penicillin, with 100,000 units of crystalline penicillin, followed by 300,000 units of procaine penicillin in aqueous solution daily, the total dose being 3,800,000 units. Nine of the 93 patients died, three being moribund on admission to hospital. Pneumococci were obtained from sputum or blood in 60 cases. All forms of treatment mentioned produced a good response. Intramuscular or oral penicillin therapy reduced the temperature quickest.

Cardio-Vascular Syphilis.

J. H. STOKES *et alii* (*The Journal of the American Medical Association*, November 3, 1951) discuss the efficacy of penicillin in the treatment of cardiovascular syphilis. They state that syphilitic aortitis must be assumed to exist in the early stages. When systolic and diastolic aortic murmurs are present, the condition is already advanced. The authors criticize treatment with iodides and mercury and with arsenicals, and they discuss the Herxheimer reaction. They state that among 111 patients with aortitis, aortic incompetence and aneurysm, penicillin caused only five mild Herxheimer reactions, and that the results of treatment generally were a great advance on those of former methods of treatment. They admit the difficulty of assessing the results. Nine million units of penicillin were used for each patient. The blood serological effects of penicillin treatment in these cases were virtually nil.

Hypertension.

C. W. FULLERTON AND I. G. MILNE (*The Canadian Medical Association Journal*, October, 1951) describe the use of hexamethonium in hypertension. Thirty-one patients were treated. Severely affected subjects with renal involvement received hexamethonium bitartrate. Doses of 250 milligrammes to three grammes were given each day by mouth. Mainly hexamethonium bromide was used. The patients were all in hospital. The results were satisfactory in all cases, including those in which renal damage and a raised blood urea level were present. Headaches and other symptoms lessened and hypertensive crises were fewer. Five young patients with very high blood pressure did not respond. When given intramuscularly the drug rapidly reduced blood pressure, temporarily. Five patients receiving the bromide salt developed malaise and a high blood bromide level, up to 80 milligrammes *per centum*. Change to the bitartrate gave relief at once. Two patients developed cerebral thrombosis and coronary thrombosis respectively.

A New Anticoagulant: 4-Hydroxycoumarin Anticoagulant Number 63.

ROYAL ROTTER, AVID O. MEYER *et alii* (*A.M.A. Archives of Internal Medicine*, September, 1951) describe the effects of a new anticoagulant, 4-hydroxycoumarin anticoagulant Number 63. The substance is allied to dicoumarol. One hundred and twenty-four patients were treated; 53 had nothing wrong with them. Others suffered from thromboembolic phenomena. Prothrombin times were checked, two milligrammes per kilogram of the drug were given, and none was given on the second day. If the prothrombin level was between 20% and 40%, 25 milligrammes of anticoagulant were given. If the prothrombin level was below 20%, no further anticoagulant was given until the prothrombin rose within the therapeutic range. If the prothrombin level was over 40%, 50 milligrammes of anticoagulant were given. The effects of the drug were apparent in twenty-four to forty-eight hours. More anticoagulant was given if the prothrombin time level was not below 40% in forty-eight hours. An average daily dose was 25 milligrammes, and this dose was maintained for twenty days when necessary. From 500 to 1000 milligrammes were given as a rule, and no toxic effects were observed. There were no erythrocytes in the urine of most patients. Gross bleeding from the nose and rectum, ecchymoses of a severe type and vaginal and conjunctival hæmorrhages were observed. Various correctives for hæmorrhage were used, the best of which was vitamin *K*, given by mouth in doses of one gramme down to 250 milligrammes. In twenty hours the prothrombin level was increased from below 20% to 70%. Blood transfusions and water-soluble vitamin *K* were of little use.

The Anæsthesiologist and the Patient with Extensive Burns.

C. R. ALLEN AND H. C. SLOCUM (*Anesthesiology*, January, 1952) present their impressions derived from 1400 administrations of anæsthetic drugs to patients with second or third degree burns. They state that their first consideration was restlessness, of which there are three causes—pain, apprehension and hypoxia. Pain was treated with an intravenous injection of morphine, eight to ten milligrammes being given in five millilitres of saline and not repeated before the lapse of twenty minutes. Respiratory depression should be prevented. Apprehension was treated by intravenous injection of "Nembutal", 100 to 200 milligrammes. The authors recommend the administration of oxygen to all severely burned patients by the best method suited to the particular conditions. Circulatory distress was treated by transfusion of whole blood in adequate amounts; it is recommended that fluids for oral administration should contain 3·0 grammes of sodium chloride and 1·5 grammes of sodium bicarbonate per litre. Respiratory distress was treated by the maintenance of a clear airway (resort being had to tracheotomy if laryngeal œdema was present) and the avoidance of medullary depression due to drugs and of tight bandages around the chest. Pulmonary œdema was treated by the "head up" position and by oxygen given under a pressure of six centimetres of water. In the early cases of this series, during the changing of dressings and excision of eschars, ordinary general anæsthetics with premedication were given, with many gross physiological disturbances of these ill patients. The authors state that starvation, dehydration, vomiting, vasomotor changes, thrombophlebitis and respiratory complications are all increased by general anæsthesia. To combat these, they have used nitrous oxide-oxygen analgesia without the patient's losing consciousness, the anæsthetist conversing with the patient throughout when dressings were being carried out. When more formidable procedures had to be undertaken, very light premedication with light nitrous oxide-oxygen-cyclopropane or ether anæsthesia was used, with surgical anæsthesia only when the grafts were actually being cut. Analgesia was best maintained by use of a semi-closed to-and-fro absorption technique.

Cortisone in Asthma.

H. S. MITCHELL AND GRACE CAMERON (*The Canadian Medical Association Journal*, April, 1952) discuss cortisone in the treatment of asthma, and evaluate their results in a study of 21 cases. Their findings are that cortisone has a definite role to play in the treatment of patients with intrinsic bronchial asthma whose emphysema is not severe enough to cause irreversible changes in their lung fields. It may be a life-saving measure in cases of *status asthmaticus* which do not respond to bronchodilators. However, the response to the drug is more or less unpredictable, and many more patients should be studied before its full value in asthma can be assessed. The drug was found to be just as effective given orally as by the intramuscular route. Dosages of 200 milligrammes for one day, decreasing to 100 milligrammes daily for several days thereafter, were sufficient. The majority of patients relapsed in from one week to three months after cortisone therapy had been discontinued, and it would appear that maintenance doses have to be continued in all cases in which response occurs to the drug. Therefore, cortisone should be tried only in those cases in which all other forms of medication have failed. Side-effects were not frequent, and one patient of this series has been taking cortisone for ten months without ill effect.

Bell's Palsy and Cortisone.

H. H. ROTHENDLER (*The Journal of Nervous and Mental Disease*, October, 1951) has treated one patient with Bell's palsy by means of cortisone. Two days after an acute onset, the patient, a female, aged fifty-nine years, was given 100 milligrammes of cortisone every six hours. Two days later control of the facial muscles began to be regained and was complete on the seventh day. An amount of 100 milligrammes of cortisone was given daily from the seventh to the thirteenth day.

The author considers that the rate of progress was unusual and ascribes it to a rapid reduction in congestion of the nerve sheath with relief of pressure. He publishes the case in the hope that others may test the usefulness of this type of treatment.

The Effect of Morphine and Pentobarbital on Ether Hyperglycæmia.

D. T. WATTS (*Anesthesiology*, January, 1952) states that ether and morphine produce hyperglycæmia by stimulating supraspinal autonomic centres with a subsequent release of adrenaline and depletion of liver glycogen; barbiturates appear to inhibit this action of ether and morphine. A series of carefully controlled experiments on rabbits is presented which illustrate that in these lower animals both morphine and ether, when given separately, produce a significant degree of hyperglycæmia. The hyperglycæmia produced by ether alone is not greater than that produced by a combination of ether and morphine. Pentobarbital had no effect on the blood glucose level of these rabbits, and ether administered after pentobarbital did not produce hyperglycæmia. The author considers that the modern trend of using a barbiturate-ether sequence in clinical anæsthesia is pharmacologically sound.

NEUROLOGY AND PSYCHIATRY.

Inheritance of Manic-Depressive Psychosis.

DAVID MERRELL (*Archives of Neurology and Psychiatry*, September, 1951) has investigated the role of heredity in manic depressive psychosis by treating statistically the reports of the Bureau of Census in the United States of America. He has found that the diagnosis of manic-depressive psychosis is made less frequently than formerly. The figures for admissions and discharges in mental hospitals confirm the cyclical nature of the disease, and there is evidence that females are more frequently affected than males. Data on twins and the high evidence of manic-depressive psychosis in relatives indicate that heredity is involved in the ætiology of the disease. The best genetic explanation for the data is that of a single autosomal dominant gene with incomplete penetrance.

Psychotherapy in Organic Disease.

LEO ALEXANDER (*The Journal of Nervous and Mental Disease*, October, 1951) discusses the element of psychotherapy in the treatment of organic neurological disorders. Nine cases are described in which definite organic diagnoses had been made; all the patients improved with active psychotherapy. The author points out that there is often an overlay which may be of hysterical, depressive or anxiety type. The diagnosis of type is important since it will determine the form of psychotherapy—hypnosis, waking suggestion, supportive, exploratory, interpretive and abreactive techniques. Some of the patients had become bedridden prior to treatment. The improvement was dramatic. The organic diseases included multiple sclerosis, intracranial aneurysm, cerebellar disease and the Parkinsonian syndrome. Due allowance is made for the possibility of spontaneous remission. In addition to the direct treatment of the emotional overlay, there is stressed the stimulation and activation of performance. Direct suggestion can have beneficial results in cases of Parkinsonism without trace of hysteria. Photographs are shown of altered gaits after hypnosis. Lastly, treatment is cited which aims at the prevention of psychosomatic reverberations. Responses to stress elicit aggravation as in arthritis. It is suggested that adrenal exhaustion phenomena may have an influence. The author sees in his results a proof of the plasticity of the organism and of the possibility that psychotherapy may mobilize reserves irrespective of the ætiology of the underlying condition.

Psychiatric Symptoms and Syndromes in Parkinson's Disease.

ROBERT SCHWAB AND HOWARD FABING (*The American Journal of Psychiatry*, June, 1951) identify four main divisions among psychiatric symptoms and syndromes in Parkinson's disease: (i) psychiatric disorders unrelated to Parkinsonism; (ii) reactive mental disturbances, usually of a depressive type; (iii) psychiatric symptoms caused by medication, for example, hyoscine, belladonna; (iv) paroxysmal psychiatric disorders probably related to Parkinson's disease, for example, anxiety attacks, compulsive thinking and counting, and paroxysmal depression associated as a rule with oculogyric disturbances. Noted also were short-lived paranoidal attacks, feelings of dissociation, paræsthesia, agitation and emotional tension, and chronic fatigue states.

Treatment of Advanced Dementia Paralytica.

PHILLIP N. BROWN (*Archives of Neurology and Psychiatry*, October, 1951) has conducted a five-year study of modes of treatment of severe *dementia paralytica*. As a result of his study he has decided that for a severe type of *dementia paralytica* encountered usually in mental hospitals, penicillin combined with therapeutic malaria is the most desirable form of treatment. He gave a course of 4,000,000 units of penicillin in dosages of 20,000 units every two hours. With this he combined a course of therapeutic malaria, which consisted of fifty hours at a temperature of 103° F. He concludes that the use of chemotherapy with this treatment probably has a deleterious effect on the course of the disease.

Psychosis in Childhood.

MILDRED CREAK (*The Journal of Mental Science*, July, 1951) states that there is no completely satisfactory definition of psychosis in childhood; it differs from conditions in the adult, since the impact is upon the immature and not yet integrated personality, thereby leading to extensive disruption of the formative patterns of personality. The mere backward child is a slower, clumsier edition of the better endowed sibling. The psychotic child is recognized almost from the onset of the disorder as peculiar and extraordinary in behaviour. The pattern is that of a schizophrenic state. One pattern of a purely psychotic type does not progress beyond a certain point, whereas in the organic psychosis with associated physical evidence of brain damage, the condition progressively deteriorates. The author personally observed 17 patients. The age of onset of psychosis ranged from one year to four and a half years. In all cases, speech was involved early and rapidly faded to mutism. Anxiety, restlessness, fear of strangers, and difficulties over sleep, feeding and elimination were commony present. The recovery rate appeared to be anything but hopeful; some children appeared to make a spontaneous recovery.

Peptic Ulcer and Personality.

CHARLES RUPP *et alii* (*The Journal of Nervous and Mental Disease*, November, 1951) studied 40 subjects of peptic ulcer by means of the cold pressor test of Hines and Brown. In this the patient rests for twenty to sixty minutes before the basal metabolic rate is determined. With one hand in ice water, the blood pressure is recorded from the opposite arm. The maximum reading indicates the response. The test was performed weekly for a year. The authors state that the response differs significantly from that of normal individuals. Two groups of hyporeactors and hyperreacters were found. The authors state that the findings are consistent with disturbance in the autonomic regulatory mechanisms. It is suggested that the symptomatology of the so-called psychosomatic disorders may result in part from this, as well as from the impact of abnormal personality and emotional constellations.

Eosinophile Cells and Electricity.

R. H. TAYLOR, M. GROSS AND I. J. RUBY (*The Journal of Nervous and Mental Disease*, November, 1951) state that eosinophile cell counts were made on 25 patients in a mental hospital. Electrostimulation with or without "Pentothal" anæsthesia produced pronounced eosinopenia usually after three hours. The authors consider that the electric current stimulates the pituitary-adrenocortical system by direct action on the hypothalamic area.

Present State of Child Psychiatry.

DAVID LEVY (*The American Journal of Psychiatry*, January, 1952) states that child psychiatry as we know it is of fairly recent origin and appears to have arisen out of three major influences, namely, the study of delinquency and the child guidance movement that developed out of it, the concept of the total personality, and psychoanalysis. The child is studied in terms of environmental pressures and intellectual capacity—work that was done by the social worker and the psychoanalyst. Multiple causations are suggested as a natural working concept of child guidance. The author states that there is much to be learnt about the psychological significance of variations in the average rate of development, in the first few years of life. Psychoanalytical therapy and practice still follow the line of the traditional adult approach, and will probably give way to direct observational studies. Play material requires more consideration in relation to its usefulness for specific types of emotional release. A more active study of drugs is needed in child psychiatry.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XXXV.

CHRONIC CONSTIPATION.

Definitions.

THE idea of constipation is a most ancient and respected concept dating back to the infancy of the race and to the infancy of each individual one of us.* It has thereby become set about with various superstitions and analogies and symbolism which makes it singularly difficult for us to translate it into the terminology of modern scientific medicine. These factors also make it next to impossible for the average sufferer to take a detached, rational and unemotional view of his affliction.

It has always been agreed that constipation is a bad thing. "Of retention and evacuation there be divers kinds", wrote Burton in his "Anatomy of Melancholy", "which are either concomitant, assisting, or sole causes many times of melancholy. In the first rank of these, I may well reckon up costiveness, which as it often causes other diseases, so this of melancholy in particular. Celsus saith 'It produceth inflammation of the head, dulness, cloudiness, headache &c.'. Prosper Calenus will have it distemper not the organ only 'but the mind itself by troubling of it', and sometimes it is a sole cause of madness."

Voltaire, most clear-headed of philosophers, describes vividly the defects in wit, character, and social grace of the habitually constipated, and by contrast the vigour, cheerfulness and merit of those accustomed to a satisfactory evacuation each and every twenty-four hours. The cult of inner cleanliness by means of the purgative, the enema, and even, in this our present century, by means of surgical extirpation of the colon, springs from an extremely deep and powerful human preoccupation. It is charming to remember that the French, with their genius for civilized behaviour, made the taking of enemata a social occasion to be enjoyed with suitable conversation and music.

It has been said that there are difficulties in translating this primarily lay idea into a scientific clinical formula. There is first a wide diversity of standards of normal defæcation between both races and individuals. The consumers of rice and mealies may expect a voluminous soft stool twice or thrice daily, while the desert nomad living on curd, mutton and dates may only need the same number of small stools per week. Neither could be regarded as a suitable standard for an omnivorous European female urban sedentary worker.

Definitions, however, have been attempted. One which has the blessing of Dr. Herbert French and Sir Adolphe Abrahams is to the effect that "Constipation is present if none of the residue of a meal taken eight hours after defæcation is excreted in forty hours". The exact clinical proof of this is obtained by giving a suitable quantity of charcoal with the meal in question and timing its reappearance. Others, however, among whom is Dr. Walter L. Palmer, will have no time factor introduced, and define constipation as "the passage of unduly hard and dry fecal material".

There is clearly a difference of expert opinion here, but even more clear is the failure in both instances to incorporate the ancient generally accepted lay meanings of the term. The patient is not concerned with his oro-anal time index, nor is he (or she) usually satisfied to be told that what he suffers from and calls constipation is really not, scientifically speaking, constipation at all. It would seem better to accept the relatively simple lay definition and keep our science for its elucidation. Constipation then means an unsatisfactory infrequency, smallness or desiccation of the stools, or any combination of these. The state of dissatisfaction may, of course, be physical or emotional. If the condition is habitual or of long standing the term chronic constipation is applicable.

Physiology.

The stomach and the colon are the expanded proximal and distal ends of the gut, possessing specialized and in some ways reciprocating functions. The stomach receives and stores at relatively long intervals quantities of foodstuffs and drink, which it chemically digests, triturates, reduces to semi-fluid consistency, and passes on over a period of many hours to the small gut. The colon at the other end receives through the ileo-caecal valve the liquid residuum after the length of the small gut has been traversed, reduces it by absorption of water to semi-solid consistency and stores it. Finally at appropriate intervals the stored fæces are propelled onward into the rectum, the defæcatory reflex is initiated, and the mass is expelled.

One of the worst vulgar errors in clinical medicine is to regard the stomach and the colon as mere muscular bags or tubes, working in mechanical, chemical and physical fashion. Each organ, on the contrary, consists of a series of highly specialized parts, working harmoniously through complicated and delicate nervous and hormonal coordinating mechanisms. Each, moreover, is under the higher nervous control of the prefrontal cortex through the hypothalamic-autonomic nerve pathways, a control which coordinates the activity of each organ with the activities of the organism as a whole, as it responds to the impositions and challenges of the total environment. Only when this is appreciated can the problems of the dyspeptic and the dyscopric become intelligible.

The fluid chyme issues through the ileo-caecal valve in jets of about ten to fifteen millilitres every half-minute or so, filling the caecum and gradually passing up the ascending colon. There is very little peristaltic or other movement in this section of the gut, and indeed the colon as a whole evinces none of the active segmentation, pendulum movements, and local peristalsis seen in the small gut. Despite this apparent lack of forcible propulsion, the chyme passes on to the transverse colon, continually losing water by absorption, until it attains the normal pasty consistency of the formed stool. The next movement is larger. Some two or three or more times in the twenty-four hours, often after the ingestion of a meal, the whole transverse colon contracts, loses its haustrations, and with a mass peristaltic wave empties its contents onward, down the descending colon to the iliac and pelvic colons, where it is usually stayed at the pelvi-rectal sphincter.

On one or more of these occasions, however, the mass peristaltic wave, particularly when the pelvic colon is loaded, carries over farther; the pelvi-rectal sphincter relaxes and the contents of the pelvic and descending colons are emptied into the rectum. This sudden distension of the latter is the normal stimulus for the defæcatory reflex, and in the normal course of affairs, if the call to stool is heeded, the voluntary acts of abdominal muscular contraction and external anal sphincter relaxation allow defæcation to proceed.

The defæcation reflex is, however, to a large degree subject to voluntary cortical control. Voluntary contraction of the external anal sphincter tends to inhibit rectal contraction, and at the same time the rectum itself tends speedily to adapt itself to the presence of its contents. Defæcation may be thus inhibited until a further mass peristalsis distends the rectum further or until by voluntary action of the individual the reflex is reinaugurated and defæcation occurs.

Most of these mechanisms described above are controlled by local gut reflexes, although, as we have seen, cortical influence enters largely into the control of the rectum and the anal sphincters. Not only the rectum, however, but the whole colon has autonomic innervations both sympathetic and parasympathetic, and these are capable of modifying profoundly the various aspects of colonic behaviour, by impulses originating elsewhere in the body or in the world outside. In general the parasympathetic influence (vagal up to the first third of the transverse colon—sacral autonomic thereafter) is motor and secretory, while the sympathetic is inhibitory, checking peristalsis and locking the sphincters. This control may give rise to a sudden complete emptying of the whole of the colon, or on the contrary, where there is a call for muscular effort all colonic activity may be shut down and the blood supply be diverted to the cardiac and voluntary musculature.

It is therefore not difficult to see how under certain circumstances and in certain types of individual, the total response to a difficult situation may include either diarrhœa or constipation of colonic origin. This is comparable with the preexamination or prematch polyuria on the one hand and the actual suppression of renal filtration during active physical exertion. It is also easy to see that even in normal individuals anxiety may produce colonic inhibition, stasis and constipation. Where such anxiety becomes chronic and poorly related to reality as in the neurotic, clearly a chronic neurotic constipation, purely by cortical-sympathetic inhibition, can be envisaged.

One of the most interesting conditions illustrating the effects of sympathetic activity on the colon is to be found in Hirschsprung's disease. Here there is a constant sympa-

thetic hypertonus involving mainly the pelvi-rectal and internal anal sphincters, leading to extreme constipation, an hypertrophied and dilated megacolon, and great abdominal distension without, however, the signs of intestinal obstruction. In some cases a spinal anæsthetic by abolishing sympathetic control allows of emptying of the enormously distended gut. The anatomical basis of the condition is in doubt.

The Causes of Chronic Constipation.

Within the colon there is a complex distribution of function. The ascending and proximal transverse portions are predominantly concerned with absorption, the sigmoid and pelvic colons have a special storage function, and the rectum is specialized for intermittent defæcation. Perhaps, as a sort of physiological ideal, we may think of the descending colon and the rectum as being normally empty for the greater part of the time, the descending colon filling only as mass peristalsis moves the bowel contents from transverse to pelvic colons, and the rectum filling to any appreciable extent only just prior to defæcation.

For healthy normal colonic function there are many necessary conditions to be fulfilled. First the colon itself needs to be anatomically normal with all its local reflex nerve arcs intact and active. Next the individual as a whole must be healthy, to the extent at least of not being subject to such gross errors of general metabolism (fever, for instance, or diabetes) which may interfere with colon absorption and motility. There must not be too much interference with local colon activity by the prefrontal-autonomic overseer. Finally, it is necessary that the colon be presented with the optimum quantity and quality of ileal chyme at one end, and receive intelligent voluntary cooperation in the workings of the rectal defæcatory mechanism at the other.

Two propositions then emerge. The first is that the actual causes of chronic constipation are numerous and diverse, ranging from *tabes dorsalis* to diabetes, from anxiety neurosis to gastric carcinoma to adolescent laziness. Secondly, whatever be the cause or causes, the effect will be one of a few simple types of chronic constipation, depending on the manner in which the colon and its function are influenced.

The Clinical Types of Chronic Constipation.

The Obstructive Type.

Constipation due to mechanical obstruction gives rise to a characteristic picture in which hypertrophy and distension of the gut and visible peristalsis are prominent. In Hirschsprung's disease in which the obstruction is due to sphincteric achalasia, constitutional disturbance is curiously small. In the true mechanical obstructions due to tumour, cicatrization, external compression and the like, the picture is that of chronic intestinal obstruction.

The Desiccative or Scybalous Type.

Stimulation to peristalsis in the colon, as elsewhere in the gut, depends to an extent on the bulk of the intestinal contents and the degree of distension of the gut wall. Now since absorption of water from the ileal chyme reduces the bulk of stools, and since the degree of absorption depends on a time factor, it is clear that any one of three factors, namely, reduction of volume of chyme, slowing of peristalsis, or increase of absorption, can set up a vicious circle in which lessened bulk, poor stimulus, slowed movement, increased absorption, lessened bulk *et cetera* become progressive. The upshot is a severe constipation with passage of small, dark, hard, "pebbly" motions, often coated with mucus from the irritated bowel wall. The chief causes of this type may conveniently be listed.

Faults in the Ileal Chyme.—The greater part of the bulk of the ileal chyme consists of the actual secretion of the digestive glands. The balance consists of bacteria, and a modicum of undigested food residue, mainly cellulose. Diminished gastro-intestinal secretion may derive from starvation; poor dietary balance, low fat content, low carbohydrate content; fever; anorexia as from gastric or liver disease; anxiety, grief, or other emotion. A low residue diet, that is to say a cellulose-poor diet, is also usually credited with reducing fæcal bulk and producing this form of constipation. The evidence for this is, however, somewhat dubious.

Excessive Absorption.—In those conditions in which dehydration occurs, excessively rapid absorption of water from the fæces may inaugurate the vicious circle. Such conditions as fevers, diabetes, uræmia, and the physiological states accompanying excessive sweating and reduced water intake are to be considered.

Slowed Colonic Propulsion.—A spastic colon with reduction of onward peristalsis occurs in a number of conditions, chiefly plumbism, morphinism, anxiety and depressive states.

The Dyschezic Type.

Dyschezia or rectal constipation is a term introduced by Hurst to denote that type of chronic constipation in which the rectal defæcatory mechanism is, by long-continued voluntary inhibition, put out of effective action. The rectum becomes merely an appendage of the pelvic colonic storage depot, with great reduction or loss of its own specific function. A normal or natural call to stool becomes rarer and rarer and the use of purgatives and enemata more and more necessary. The rectum is never emptied of its load of hard dry fæces except for brief intervals following purgation.

The causes are primarily psychological—laziness, ignorance, false modesty are largely blamed. Freudian psychology has several deep and dark interpretations involving such concepts as ano-erotic narcissism, gold-symbolism, and suppressed antiparental aggressions. Occasionally an organic basis is found such as poor abdominal musculature, or destruction of the reflex arc as in tabes or other nervous lesions.

The Asthenic Type.

Asthenic constipation is described as occurring along with colonic muscle and mucosal atrophy in old people, in the obese, in chronic diverticulitis, and in life-long consumers of purgatives. It is possible that it represents a true entity in which the functional basis is failure of mass peristalsis. It is, however, also thought to be merely the end result of long-continued dyschezia. It is characterized by constant filling of the whole colon with dryish fæces, only with great difficulty removed by enema or purgative.

Diagnosis and Treatment.

The cause of chronic constipation, it is apparent, may lie, and must be sought, in any and every nook and cranny of the individual, or even in his environment. The colon is in many instances, perhaps in most, the mere sounding board of discord elsewhere, the unfortunate whipping boy for organs and functions thought to be beyond suspicion. The amount of coercive local therapy directed at the colon (as also at its unfortunate sister organ the stomach) probably represents the grossest misdirection of human therapeusis in history, even allowing for the phlebotomists.

The only remedy is to refuse to treat the word constipation. Our every diagnostic resource must be turned instead on the individual complaining of this age-old woe. An accurate history of the patient, as well as of the particular dissatisfaction that he or she terms constipation, is vital. Next must come accurate and complete clinical examination, never by any chance omitting (need it be stressed?) digital examination *per rectum*. Later, if necessary, resort may be needed to proctoscopy, sigmoidoscopy, radiography and the like.

Only thus can one disclose or discount the presence of nervous, psychiatric or metabolic anomalies or local disease in far-removed organs. The anxiety breakdown, the "little stroke" that Dr. Alvarez describes so well, the early tuberculosis of the lung, or duodenal ulcer, the pituitary depression after pregnancy or the thyroid depression after the menopause, may each and all be causative.

Such matters dealt with, the questions of dietary habits, and of defæcatory habits and habitus, may offer the clue, or the less ponderable factor of constitutional make-up. The thin, bright, alert young woman who combines her constipation with dysmenorrhœa of spastic type suggests a constitutional sympathetic hypertonia; the obese and flabby depressive is the natural prey of dyschezia.

In treatment one must, if one cannot remove the cause, alleviate the symptom. In constipation with its curious infantile emotional aura this is particularly so, but it must be realized that one is dealing with a dissatisfaction and a discomfort. Purely functional constipation has never done anyone any harm whatever except to "the mind itself by troubling of it". But this is a very strong reason for ensuring that even symptomatic treatment is efficient.

The remedies of antiquity, the purgative and the enema, have a small and minor place still in treatment. The treatment of dyschezia requires reeducation of rectal function—first the patient must be enlightened and contrite—then the enema may be a useful adjuvant used regularly for a few

days or weeks till the sensitivity of the rectum and habit training are restored. There is no place for violent purgation in the treatment of chronic constipation, but in the elderly patient with the asthenic type of disorder such muscle stimulants as senna infusion or phenolphthalein may be unavoidable.

To use either purge or enema, however, in the treatment of the desiccative type is useless. The object here is to increase fæcal bulk and restrain desiccation to the stage at which sufficient bulky stools produce efficient bowel stimulation and motility. Here agar-agar, psyllium, vegetable mucins and the like have their place, and more particularly the correction of dietary indiscretions. To treat these people with enemata is as stupid as to treat the dyschezics with high cellulose diet. Where nervous influences or abnormal psychological reactions are discernible, sometimes mild sedation and readjustment of emotional balance may help. At least, however, it is usually possible by explanation of physiological mechanisms to relieve the anxious of the additional anxiety that his constipation is a sign of dread and perhaps mortal import.

H. R. LOVE,
Brisbane.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 26, 1952, in the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. R. H. MACDONALD, the President, in the chair.

Painful Shoulder.

DR. RICHARD HODGKINSON read a paper entitled "Painful Shoulder" (see page 296).

DR. S. G. NELSON read a paper entitled "Painful Shoulder" (see page 293).

DR. N. H. MORGAN said that the two papers, if taken together, were completely comprehensive in subject matter. He was interested in the shoulder-hand syndrome, and would have liked to hear more on that. It was more important than some of the other conditions that had been mentioned, because it was more common and more intractable, and was associated with more pain and disability. His attention had been drawn to it by a recent case. A woman had sustained a lesion of the supraspinatus tendon, and developed the complete picture of Duplay's syndrome. Dr. Morgan wondered whether the speakers agreed that periarthritis of the shoulder was a special variant of the shoulder-hand syndrome, so called by Steinbrocker in 1937. Dr. Morgan said that at one time in operations on tendons he had transplanted the long head of the biceps to the coracoid process in the treatment of frozen shoulder. The operation did not effect any material improvement. Dr. Morgan thought that the pathological background was quite different. Neviaser had recently operated in ten such cases, and had tried to discover the essential pathology. He stated it to be fibrosis of the undersurface of the capsule; the loose flap of capsule that must exist there if the arm could be abducted became thickened and adherent to the undersurface of the humerus. Cure could be effected by manipulation or by the use of a blunt dissector. If that was a late stage, the area being very dry and effusion absent, there must be an early stage. However, post-mortem examinations had not been carried out on subjects having the condition in an early stage, so no certainty was possible. The only reference was to work reported by Leriche, when he was performing periarterial sympathectomy for the early stage of frozen shoulder. Leriche's observations led to the conclusion that the underlying condition was a vasomotor reaction of some type. Dr. Morgan had seen the condition follow *herpes zoster*. The Liverpool school was against manipulation in the treatment of frozen shoulder in the early stages or when pain extended below the elbow. Sometimes heat was of help, sometimes cold; but often the pain was resistant. He had known two cases in which manipulation was carried out early, and an acute vasomotor reaction resulted. Thickening of the palmar fascia was often found if looked for. He had found it in two cases in the out-patient department that afternoon. He had seen such a vascular reaction follow too early manipulation. It seemed more a parasympathetic than a sympathetic dystrophy and resembled Sudeck's atrophy. The whole problem was what they could do to stop the pain early in the piece and save the patient from going through the full cycle for months. He would like to hear something about the use of tetraethylammonium bromide (TEAB), cortisone and ACTH. He also wanted to know whether the speakers believed that trauma played a part in the ætiology of the syndrome.

DR. A. F. DWYER referred first to acute calcification of the shoulder. He said that on one occasion, to relieve severe pain, he had operated and removed the calcium. He also removed some of the surrounding tendon and of the normal tendon nearby, with the object of investigating the phosphatase content. He had found that the phosphatase content of the tendon immediately surrounding the deposit of calcium was much increased. Nothing was to be learnt from the general blood chemistry, and it was not known what part increased phosphatase content played in acute calcification. Those who had such attacks must have some tendency to it. Dr. Dwyer agreed with Dr. Morgan's remarks concerning frozen shoulder. He said that he had had some experience with cortisone in the treatment of stiff shoulders. One patient with true Sudeck's atrophy for six months had never been able to lift the hand above shoulder level. After twenty-four hours of cortisone treatment all pain had gone from the shoulder, in two days the hand could be raised above shoulder level, and at the end of a week all shoulder movement had been restored. In such a case of Sudeck's atrophy in the exudative stage, manipulation would have been useless. Dr. Dwyer then referred to a patient with Sudeck's atrophy in the hand; fibrosis was present affecting the palmar fascia, tendon sheaths and joints, but there were no contractures; there was radiological evidence of atrophy right up to the shoulder joint. She was treated with cortisone for one month, and then with ACTH. One reason why treatment was carried on for so long was that she came from the country. Shoulder and hand movements returned fully. Similar treatment had been tried in a case of median neuritis with similar vasomotor changes in the hand and also a stiff shoulder; cortisone relieved the patient from pain, and no contractures occurred, but full shoulder movement did not return. Two other patients treated with cortisone by other clinicians had done well. The mode of action of cortisone was akin to its action in allergic conditions. Dr. Dwyer said that not nearly enough attention had been paid to Lewis's paper published in 1936, when he found that stimulation of the skin of sufficient intensity to cause pain would be followed by hyperalgesia and a sensation of burning that would last up to twenty-four hours. It was due to an axon type of reflex. There was no need to worry about de Nó's hypothesis, because Lewis had proved what could happen. What happened in the skin would almost certainly happen in deeper tissues. Cortisone seemed to have an effect in inhibiting the formation of substances liberated in the axon reflex. Inflammation might be a neurovascular manifestation of trauma, and it could occur without trauma. Cortisone simply suppressed the vascular reflex.

DR. R. READER said that it was refreshing that both speakers had kept their explanations to conditions of demonstrable pathology; no reference had been made to fibrositis or other intangible and unlikely conditions. Dr. Nelson had summarized the structures in which pain occurred as joint tissues, nerves and remote organs giving rise to referred pain, and had also implied that pain might occur in vessels through reflex sympathetic mechanisms. The internuncial pool theory had been invoked in explanation; Dr. Reader described three cases to illustrate the occurrence of pain in the extremities associated with sympathetic disturbance. The first was in a middle-aged woman who suffered very severe pain in both hands, more in the left than in the right; there was thickening of the fingers, and the diagnosis of rheumatoid arthritis had been made. The fingers were red and swollen, and the condition seemed more a vascular than a joint condition. She was treated with a ganglion-blocking agent, "Priscol"; it did not relieve the pain, but produced Horner's syndrome on the side on which the pain was more severe. A second patient had complained of the same type of pain; the right side was affected, and her fingers were swollen, thickened, red and warm. She also had Horner's syndrome on the affected side, complaining of lack of sweating of the right side of the face and the right arm; no cause for the sympathetic disturbance could be found. A third patient, a man, had sustained a back injury as a result of a fall from a tractor. His symptoms were fairly typical of lumbar disk protrusion. As well, the foot on the affected side was warmer than its fellow. On exposure of his lower extremities to the cold of room temperature, 18° C., the temperature of the

sound limb fell normally, but the temperature of the affected limb rose slightly to 33° or 34° C. This response was typical of lumbar sympathetic paralysis. Dr. Reader said that he would be interested to know the experience of orthopædic surgeons in this type of lesion. Dr. Nelson had handed over the cervical disk to the surgeons. Dr. Reader did not know whether they would accept it. Dr. Gilbert Phillips had recently talked to workers in Oxford, notably Ritchie Russell, about cervical disk lesions with pressure, and they had pointed out that the cervical part of the cord was fixed to the spinal column in three places (by the *ligamentum denticulatum* and the anterior and posterior nerve roots), and it had been possible to show areas of gliosis corresponding to those three places. The symptoms seemed to be related to the amount of mobility in the cervical part of the spine, and that fact supported the present practice of immobilizing the cervical part of the spine in some form of collar, rather than resorting to operation.

DR. W. D. STURROCK expressed his disappointment. He said that he had come to the meeting hoping to have some of the perplexing and common problems answered. He had heard a very discursive talk on the causes of painful shoulder. It was a common condition, and they had all hoped to make some progress in the knowledge of what should be done. Dr. Nelson had given a long list of causes; Dr. Sturrock disagreed with some of them. He had never seen a cervical rib causing pain in the shoulder; it was hard to see why it should. Referring to acroparæsthesia, Dr. Sturrock said that it was common; it came on at night, with swelling of the wrist and hand, but not with pain in the shoulder. Painful shoulder meant some degree of trauma. It followed simple fractures, Colles's fractures and so on. Because attention was not paid to full immobilization of the shoulder, stiffness of the shoulder followed, and became very intractable and difficult to deal with. Dr. Sturrock said that he had hoped to get some information on ganglion-blocking drugs, cortisone, ACTH and para-aminosalicylic acid. The affected middle-aged women gave a history of some minor injury. Many of them had slipped and fallen, and put out their hand to save themselves. Often the accident had happened a couple of months earlier. Examination revealed that the range of abduction was about 60°, and that very little rotation was possible. Such patients presented every one of the rare types of pain in the shoulder, and they were the ones to whose treatment it had been hoped that a clue would be given. In many cases the pain was due to direct trauma to the joint, its capsule and the surrounding tendons. Dr. Sturrock made a plea that treatment should not be continued for months and months. He had had an interesting case referred to him. The patient was a woman who had sustained a mild injury to her shoulder. She received varying forms of therapy over a period of nine months, and was rather worse than when she began; then surgery gave her relief. At operation he removed the acromion process; this was not often indicated, but had produced rapid relief. Dr. Sturrock said that he agreed with Dr. Morgan that in many cases of painful shoulder with stiffness due to some sympathetic disturbance, decalcification of bone went on and the condition remained one of Sudeck's atrophy in the distal parts of the extremities. That was the problem they faced in any nine out of ten cases. At the meeting they had not advanced at all in the common problem of painful shoulder.

DR. JAMES ISBISTER said that he had seen two or three patients suffering from the shoulder-arm syndrome. It had been suggested by an orthopædic surgeon that they should use cortisone in treatment. It was not surprising that ACTH or cortisone should be used in any intractable condition, and in the cases quoted it appeared to have had very dramatic effects. In cases in his experience some improvement followed its use, but that might have been due to intensive physiotherapy. Dr. Isbister asked whether Dr. Nelson would say that the condition under discussion was a form of the adaptation syndrome. Dr. Isbister said that he saw many patients suffering from acroparæsthesia. One patient whom he saw frequently was now quite better; she had had the typical syndrome in both arms. Her occupation was packing butter in boxes in a factory; the bench at which she worked was very high, and she was obliged to work all day with both arms in the abducted position. Dr. Isbister suggested to her that she have the bench lowered or use a small stool, whichever was the more convenient. She used a small stool, and the syndrome disappeared. Dr. Isbister wondered how such a position of the arms could cause acroparæsthesia.

DR. E. HASLETT FRAZER referred to the use of cortisone. He said that he had never been an enthusiast for it. In 1951 he had been in Rochester, and Slocumb and Hench were emphatic about the value of the use of 100 milligrammes of cortisone per day for seven days in the treatment of painful shoulder. The severity or otherwise of the condition made no difference. Then treatment was continued with 50 milligrammes per day for six weeks accompanied by physiotherapy. Good results were claimed. Dr. Frazer was not sure about that; he had not had the opportunity of seeing any good results. On the other hand, Steinbrocker in New York was not keen on the use of cortisone in this condition. Instead he was suggesting the injection every day for four days of "Butapyrin" (phenyl butazone) as a sedative and antispasmodic. Dr. Frazer said that he had tried the method a couple of times with some success. But when Dr. Nelson talked about the "life situation" being difficult, Dr. Frazer agreed that such a condition did add very greatly to the intensity of the pain. Dr. Hodgkinson had shown interesting photographs of shoulder joints of cadavers. They, of course, would appear to militate against any nervous factor, but they were cases of long standing. After thirty years he was just as doubtful about painful shoulder as he had been before. He had one himself, which was due to his having fallen down some steps years earlier. He had had all kinds of treatment, but he still had a painful shoulder. But he had noticed that if he became worried or had an attack of influenza, the pain became very much worse. If he felt well, he could go out and chop wood for a couple of hours without pain. He did not know what the condition was—it might be a torn capsule. He did not think that there was the slightest doubt that some of these varying sensitivities to pain were the result of unconscious worries or emotional distress, or a "difficult life situation". At the moment he thought that the best treatment was the application of an abduction splint for six weeks, but he had never yet seen a patient who would submit to it. Some benefited by heat, some did not. He had had manipulation, massage and injections of lipiodol around the shoulder for many years. He thought that painful shoulder was often due to trauma and early periarthritis, and often no X-ray changes were obvious. One of the most interesting approaches to the painful shoulder which he had seen was that of James Cyriax, of Saint Thomas's Hospital, London, whose differential diagnosis and manipulative treatment were worth studying. A descriptive film of his work was probably now completed. It was instructive and timely to hear the comments of the speakers on the subject. Out of some 200 papers given at the recent Barcelona Congress he had not heard, or known of, more than one dealing with the shoulder joint. Dr. Frazer said that he would like to hear a few words on treatment; the patient consulted the clinician in order to get well, and while discussion of the diagnosis was necessary, the patient was chiefly interested in being relieved of the pain.

Dr. Hodgkinson, in reply, said that he was still as confused as everyone else about the problem of treatment. In answer to everyone's question, he would say that if they knew the cause of the pain, they should try to remove it. There were certain causes; both Dr. Nelson and he had mentioned some of them. If the pain could be removed, then activity was possible, and a stop was put to the pathological change that occurred in the shoulder-hand syndrome. Those pathological changes had been well described in de Palma's book. De Palma talked about all the small structures that became gummed up with exudate and tied up together. Probably that was a reaction to stress, and the bone changes occurred with disuse. It was possible to stop the process if the patient could be induced to move the shoulder. Decalcification occurred if a child lay on its back for a long time in a frame—in other words, it occurred with disuse. Dr. Hodgkinson also thought that in many cases serofibrinous exudate occurred with disuse. It was necessary to assess the susceptibility of the patient to pain in order to be able to get him to move. Early movement was one lesson in treatment that he could suggest.

Dr. Nelson, in reply to Dr. Morgan's question about the pathology of the shoulder-hand syndrome, said that the original paper by Steinbrocker described three stages of development, and it appeared that the condition was a fairly well-defined entity. The concept of the shoulder-hand syndrome had been widened, and the cases varied a good deal from those following Steinbrocker's picture fairly closely to those in which the picture was incomplete. For example, the shoulder part might predominate in some, in others there might be a little thickening of the palmar fascia and vasomotor changes in the hand without the full picture. One patient had thickening of the palmar fascia in the region of the fourth metacarpal bone. That patient had had two accidents, one about nine months previously, and another while she was awaiting admission to hospital. There was not much residual neurological change left. She had only slight palmar fascial thickening, but she had a

good deal of pain and limitation of movement of the shoulder. Young and Pearson, in their article published in The Medical Journal of Australia of June 7, 1952, had two photographs. The first showed a coronal section of a normal shoulder joint, and the inferior part of the capsule could be seen hanging down in a fold for half an inch below the inferior margins of the articular cartilages. The second showed a coronal section of a shoulder from a patient who had suffered from the shoulder-hand syndrome; the inferior part of the capsule was thickened and stretched tightly between the edge of the glenoid fossa and the neck of the humerus, to which it was adherent. Dr. Nelson said that he had often thought that the changes were secondary, rather than primary. He had seen manipulation of the shoulder followed by hand symptoms. A recent article had appeared in *The Journal of the American Medical Association* on the reduction of pain in osteoarthritis of the hip by the use of cortisone; it was given, not so much to cope with the condition of stress, as to act in some way as an analgesic. Dr. Frazer's comments on the American experience had been interesting. Dr. Nelson knew that cortisone had been used in Sydney with considerable success. One wondered whether the improvement would last when the effect of the cortisone administered had passed away; often one found that relief ceased some little time after cessation of therapy. Dr. Nelson went on to say that he had not used TEAB, only TEAC, but that had afforded considerable relief of pain. Referring to the mechanism of decalcification, Dr. Nelson said that disuse was a well-known cause, and also an increased blood supply. It was significant that in the early stage of the shoulder-hand syndrome there was a phase of increased blood supply and heat, which appeared to be a manifestation of Lewis's nocifensor mechanism. It was odd that the condition should be connected with vasodilatation and not with vasoconstriction. That was why it was difficult to explain it in the light of present knowledge of the anatomy of the autonomic nervous system. Dr. Nelson knew nothing about the phosphatase content; he had been interested in Dr. Dwyer's comments. In reply to Dr. Sturrock, Dr. Nelson said that Dr. Sturrock saw fewer neurological conditions; he (Dr. Nelson) saw a number. He was basing his remarks on his own personal experience. It was possible, but not invariable, to find pain in the shoulder in the lesions mentioned. In reply to Dr. Reader, Dr. Nelson said that he believed that the paralysis of the sympathetic which he had mentioned in the three cases would be an alternative explanation for the vasodilator mechanisms involved. Dr. Isbister had introduced the question of stress as a factor in the ætiology. Dr. Nelson said that it was very difficult to be dogmatic about it. Certainly the patients mentioned had been under a good deal of stress, but they did not fulfil Selye's requirements for patients suffering from the exhaustion phase of the diseases of adaptation. It was difficult to summarize the treatment in a few words; Dr. Hodgkinson had covered both aspects of the question by saying that the main thing was to make an accurate diagnosis, and the treatment would arise out of that.

Dr. Macdonald, from the chair, thanked the speakers for their papers. He said that it was a matter of regret that so few members of the Branch had attended the meeting to hear papers on such an interesting subject. He assured the speakers that their papers would be read widely and by a much greater number.

A meeting of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on April 17, 1952. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital. Parts of this report appeared in the issues of August 2, 16 and 23, 1952.

Intrathoracic Goitre.

Dr. F. F. Rundle showed a married woman, aged forty-one years, who had noticed a swelling in the neck for six or seven years. More recently she had become unduly irritable and excitable, with occasional palpitations and a feeling of suffocation and pressure in the lower part of the neck. On examination of the patient her colour and state of nutrition were normal, and there were no clear signs of hyperthyroidism or hypothyroidism. A deeply placed goitre involving both lobes and isthmus was palpable low down in the neck. Radiological examination revealed a smooth rounded mass projecting downwards and backwards from the root of the neck into the right upper part of the thorax. It moved freely upwards on swallowing and displaced the trachea and œsophagus slightly to the left and forward. The patient was admitted to the Wakehurst Wing, and at operation on August 28, 1951, through an extended collar incision, the cervical part of the goitre was fully mobilized and subtotally resected. A stalk of goitrous tissue was found extending from the middle of the posterior aspect of the right lobe downwards and backwards into the thorax behind the bifurcation of the innominate artery and the first parts of the right subclavian and common carotid arteries. Postero-laterally lay the inner border of the first rib, and on the inner side were the œsophagus posteriorly and the trachea anteriorly. The right recurrent nerve ran upwards and medially on its anterior surface. At a subsequent operation on October 9 the right side of the thorax was opened through the fourth intercostal space, and this stalk of tissue was found to be continuous below with a mass about the size of a grapefruit situated in the posterior and superior part of the mediastinum. The intrathoracic mass was resected *in toto*. X-ray films and sections of the tissues removed were shown at the meeting.

Another patient of Dr. Rundle's, a man, aged fifty-two years, had developed "virus pneumonia" about the middle of 1951 while in Malaya. Recovery was very slow and was apparently complicated by some collapse of the left lung. Otherwise there were no symptoms. Examination of the patient revealed the presence of a deeply placed goitre, apparently larger on the left side and extending into the thorax. X-ray examination showed a mass which extended down at least to the level of the neck of the fourth rib and which moved freely upwards on swallowing. The trachea was displaced well over to the right. The patient had been admitted to Wakehurst Wing for surgical treatment. Dr. Rundle discussed the problem of the operative approach to substernal and intrathoracic goitres and said that it was being described in detail elsewhere.

Unilateral Lid Retraction with Minimal Thyrotoxicosis.

Dr. Rundle's next patient, a single woman, aged nineteen years, had noticed the left eye prominent for the past six weeks, but had no other symptoms. Examination of the patient showed the clinical features characteristic of retraction of the upper lid in Graves's disease. The retraction was due to spasm of the striated *levator palpebræ superioris*, and the clinical evidence for this was demonstrated. The thyroid gland was not palpable, but the basal metabolic rate was +26%. Dr. Rundle said that an interesting point was that though the left eyeball was only 1·5 millimetres more prominent than the right, the patient had been thought in another clinic to have an orbital tumour, and for some time Graves's disease was unsuspected.

Suspected Seminoma of the Testis.

Dr. Rundle's last patient, a man, aged forty-two years, had been referred for surgical treatment by Dr. Douglas Anderson. The patient's history was that he had felt quite well until three weeks before admission to hospital, when he developed dull aching pain in the upper part of the abdomen; it was worse at night and prevented him from sleeping. A week before admission to hospital, for a period of twenty-four hours, his pain was very intense, and he passed three tarry black motions. He felt nauseated but did not vomit. He had not lost weight. Six months previously he had developed an acutely painful swelling in the left side of the scrotum, for which he had been admitted to the Repatriation General Hospital, Concord. The swelling and pain had rapidly responded to sulphonamides and he had been discharged from hospital with the diagnosis of acute epididymo-orchitis. The patient was a healthy looking subject with no wasting or pallor. A large firm mass in the right hypochondrium extended also into the epigastric, umbilical and right lumbar zones. Pre-operative radiological examination showed the thorax, œsophagus, stomach, duodenum and colon to be normal. The abdominal mass was displacing the right kidney outwards and backwards. At operation by Dr. Alban Gee on October 16, 1951, the left testis was first explored and bisected. Nothing abnormal was found apart from a small collection of clear fluid in the *tunica vaginalis* sac. Both testes were of normal size, and the cut surface of the left testis appeared healthy. The abdominal mass was then explored through a right subcostal incision, exposing the kidney posteriorly, and extending into the peritoneal cavity in front. A very large mass was found above and medial to the kidney. The duodenum was stretched out over it and displaced to the left. It was very firmly fixed and widely infiltrating the retroperitoneal tissues behind the pancreas and round the inferior *vena cava*. Snippets were taken for microscopic examination, but removal of the tumour was deemed impossible. Dr. C.

Graham submitted the following microscopic report: "These sections show masses of malignant tumour cells separated by strands of fibrous tissue. The cells are fairly uniform in size, they are round, and the margin of the cytoplasm is ill defined. A fairly thick layer of fibrous tissue separates the tumour from the kidney tissue which appears normal. The cells are unlike those of a renal carcinoma. Their appearance and arrangement is strongly suggestive of a seminoma. Although I am assured that on clinical grounds this diagnosis is extremely unlikely, I can think of no alternative. However, there is no doubt that this is a malignant tumour and that it is carcinomatous rather than sarcomatous in nature."

Dr. Rundle said that the patient made a good recovery from the operation, but ten days later developed rapidly progressive œdema of both lower limbs, especially the right. After intensive radiotherapy, given by Dr. Harold Ham, the œdema of the patient's lower limbs and the intra-abdominal mass completely disappeared, he gained weight and he lost all his symptoms. At a recent follow-up examination he had complained of some cough and dyspnœa on exertion, and radiological examination of the thorax revealed a large opacity in the region of the left lung root. A further course of X-ray therapy was given for that. Both testes appeared normal, and there was no recurrence of the intraabdominal mass. Dr. Rundle commented that the location of the mass in the paraaortic region, the microscopic report, and the rapid response to radiotherapy suggested that the condition was a seminoma. He wondered if it was arising in a Wolffian ridge remnant.

Tuberculous Empyema.

DR. C. G. BAYLISS showed a male patient, who in April, 1951, when he was aged thirty years, had become ill with an acute respiratory illness diagnosed as pneumonia and pleurisy. On May 20 he was admitted to Canterbury District Memorial Hospital, where he was found to have a large pleural effusion on the left side. Aspiration of the pleural cavity was performed on a number of occasions. Acid-fast bacilli were recovered from the pleural fluid. No acid-fast bacilli were found in the sputum. The Mantoux test result was positive. In spite of repeated aspiration the lung could not be made to expand and remained at the same degree of collapse. It was decided after three months' observation that the lung was probably unexpandable, and on September 8 the patient was transferred to the Royal North Shore Hospital of Sydney for further treatment. Further aspirations of the chest were carried out without further expansion being achieved. Bronchoscopy revealed no abnormality. On October 11 decortication of the lung was carried out by Dr. M. P. Susman. The layer of organized exudate "peeled off" the lung was one-quarter of an inch thick. Expansion of the lower portion of the lung was achieved, but not full expansion of the apex. The post-operative course was complicated by hæmorrhagic oozing. An effusion developed, and this gradually became purulent. On November 20 drainage of the empyema was carried out. On December 18 a first stage thoracoplasty was carried out by Dr. Susman. Dr. Bayliss said that since then obliteration of the pleural cavity had gradually taken place, and when the patient left hospital on April 6, 1952, the capacity of the sinus was only 10 cubic centimetres. The general condition of the patient was excellent, and complete stabilization was confidently anticipated. In summing up, Dr. Bayliss said that the patient appeared to have had a primary tuberculous infection of the lung accompanied by pleural effusion. Unlike that in the majority of such cases the effusion had failed to absorb, and an unexpandable lung had followed. Decortication had enabled partial expansion of the lung to be achieved. Convalescence had been complicated by empyema which required thoracoplasty for its control.

Chronic Lung Abscess.

DR. IAN MONK showed a boy, aged five years, who had been admitted to hospital in July, 1951. One year before he had had an attack of pneumonia, which was treated with a course of sulphadiazine. Recovery was not complete in that cough persisted. A month after this attack a further respiratory infection occurred, with exacerbation of the cough, and high temperature, necessitating admission to hospital and treatment with penicillin. In October, 1951, a severe hæmoptysis occurred, and the patient was admitted to the Royal Alexandra Hospital for Children, where further hæmoptyses necessitated a blood transfusion. The patient was in this hospital for seven weeks and was discharged, well. Chronic cough recurred with offensive sputum, and a further pneumonia-like attack led to admission to the Royal North Shore Hospital of Sydney. A history was elicited from the parents that prior to the original attack of pneumonia the patient was said to have swallowed some grass, choked and had a fit of coughing. No foreign body was ever actually seen by the parents. Examination of the patient revealed a normally developed, healthy looking child. The breath was noticeably offensive and a partially suppressed cough was present. Grade III clubbing of the fingers was present. Crepitations were audible over the lower lobe of the right lung. A plain X-ray examination revealed "pneumonitis" involving the lower lobe of the right lung with the presence of a small cavity. A bronchogram revealed saccular bronchiectasis of the three basal divisions. Bronchoscopy revealed offensive mucopus coming from the lower lobe of the right lung. After seven weeks' preparation, a right lower lobectomy was performed. Post-operative convalescence was uneventful. The patient had since remained well, and was a normal child clinically, and practically normal radiologically.

Urological Conditions.

DR. COLIN EDWARDS showed a series of X-ray films to illustrate interesting urological conditions, especially renal tuberculosis associated with pulmonary tuberculosis.

Hypertension with Unilateral Renal Disease.

DR. ALBAN GEE first showed two patients suffering from hypertension in whom the pathological state of one kidney was thought to be the basis of the condition. The first patient, a man of forty-seven years, had been referred because of repeated headaches and the presence of albumin in the urine. He had had a stone removed from his right kidney twenty-three years before, and his blood pressure was 170 millimetres of mercury, systolic, and 110 millimetres, diastolic. An excretion urogram demonstrated a stone the size of a shilling in the pelvis of a small right kidney, the concentration of drug being poor. The left kidney was larger than normal. At cystoscopic examination a good indigo carmine test result was obtained from the left kidney, but no dye was seen after twenty minutes from the right. A right pyelogram confirmed the presence of a stone in the pelvis of a small kidney, and a nephrectomy was performed. The pathological report stated that most of the glomeruli were sclerotic and functionless amidst interstitial fibrosis; it was a picture of advanced chronic pyelonephritis. A month later the blood pressure had fallen to 126 millimetres of mercury, systolic, and 84 millimetres, diastolic, and had remained at this level one year later.

The second patient was a man, aged twenty-six years, who had reported in 1949 with right renal colic occurring in attacks over the previous six months. Investigation revealed a large hydronephrosis of the right kidney, with a blood pressure of 165 millimetres of mercury, systolic, and 100 millimetres, diastolic. Operation was performed in January, 1950, and it was recorded that a dilated renal pelvis was found, there being only a small amount of renal tissue. Adhesions around the lower pole and an aberrant artery were divided, and a nephrectomy was performed. Two years later an excretion urogram revealed a normally functioning left kidney, but no excretion from the right kidney. At the time of the meeting, two and a half years after the operation, the patient had slight tenderness over the right kidney, and a blood pressure of 190 millimetres of mercury, systolic, and 110 millimetres, diastolic. At a cystoscopic examination, a pale blue colour only appeared in the urine from the right kidney following the injection of indigo carmine. A retrograde pyelogram showed the right kidney to be small and contracted, though the pelvis still showed some dilatation. The urine was uninfected. Dr. Gee said that the patient had been recommended for admission to hospital for right nephrectomy, in the hope that although his hypertension had been present for over two years, removal of the kidney would lead to a lowered blood pressure. He was a young man with progressive hypertension and a unilateral diseased kidney, and it was felt that no harm, and indeed much benefit, would probably ensue. The condition appeared to be one of chronic sclerosing or atrophic pyelonephritis, which might have led to renal ischæmia, and to resultant hypertension. That was almost certainly the case in the first patient presented, and was the ideal pathological state in which to expect a favourable result from nephrectomy.

Renal Calculus in a Child.

Dr. Gee next showed a child, aged five years, who had been referred by Dr. O. M. Spence, of Wyong, with abdominal pain and a suggested diagnosis of nephritis. There was a past history of "pyelitis" two years previously, and investigation of the urine resulted in culture of coliform organisms. Calcium oxalate crystals were also seen in the urine. The

patient was admitted to hospital for investigation, when an excretion urogram revealed a calculus in the left ureter at the level of the lower border of the sacro-iliac joint. There was early dilatation of the collecting system above. Cystoscopic examination found an obstruction one inch up the left ureter, beyond which no instrument could be passed, and a meatotomy was performed. Several short attacks of ureteric colic then occurred, until about seven months from the first visit, when the patient passed the stone. It proved to be composed of calcium oxalate. Nearly one year later the patient returned with further pain of left ureteric distribution. Her urine again yielded a culture of coliform organisms. Radiographs showed that a further calculus had formed and was at the level of the left ischial spine. It had moved down to the uretero-vesical junction by the time cystoscopy was performed, when the ureter was dilated with Brasch bulbs without difficulty.

Hydronephrosis with a Non-Functioning Opposite Kidney.

Dr. Gee then presented a woman, aged forty-six years, who had been referred because of attacks of pain in the left loin ever since a hysterectomy had been performed seven years previously. Pain had been first noticed while she was in hospital after this operation, and was at first of a rather constant type. More recently she had developed frequency of micturition during the daytime, though her nights were undisturbed. An excretion urogram revealed that the left renal pelvis was considerably dilated, and the calyces also showed signs of back pressure. The appearance was consistent with upper ureteric obstruction, most likely from the pressure of an aberrant vessel. There was, however, no excretion at all from the right side. Cystoscopic examination was performed, and a normal but delayed excretion of indigo carmine was obtained from the left side. No instrument, however, could be passed more than three centimetres up the right ureter, and no urine could be observed or obtained. A similar result was obtained on each of two subsequent examinations, once by another urologist. Dr. Gee said that the patient was at present awaiting admission to hospital for operation on her left or sole functioning kidney. It seemed highly likely that the right ureter had suffered trauma in the classical area just above the bladder, during her pelvic operation, and that the increased strain on the left kidney had accentuated its pathological state.

In discussing the case, Dr. Gee remarked that injury to the ureters was much commoner in pelvic surgery than had been realized. That was due to the fact that although the severed ureter would eventually cause urine to be extravasated, the ureter that had been merely included in a ligature was at once occluded and its kidney ceased to function. Some pain might be felt in that loin, but often the patient experienced no symptoms, except perhaps a slight ache in the opposite kidney, which had hypertrophied in compensation. The remedy for that was the insertion of ureteric catheters prior to any operation in the pelvis, when distorted anatomy was present. It was felt in the present case that the history was too long for repair by open operation to be attempted, and that the main efforts should be directed to giving the patient one reasonably stable kidney.

Tuberculosis of Right Sacro-Iliac Joint.

DR. A. R. HAMILTON presented a man, aged twenty years, who in August, 1947, had first noticed pain in the lower portion of his back radiating down his right leg. He had a family history of pulmonary tuberculosis and was having chest X-ray examinations at six-monthly intervals. On admission to hospital, two weeks later, he was found to be febrile and was tender over the right sacro-iliac joint. Abduction of the right hip was limited, owing to pain, but all other movements were freely carried out. Investigations showed he was Mantoux-positive, and a certain amount of bone destruction around the right sacro-iliac joint was noted in the X-ray film. His chest was radiologically normal. His blood count was normal, but he had a blood sedimentation rate of 20 millimetres in one hour. He was placed on a plaster bed and given penicillin for six weeks. His temperature remained elevated for several months, but then gradually subsided. Progress was slow but steady. His appetite increased, and he began to put on weight. In April, 1948, he was given calciferol, but this upset his stomach and was then discontinued. During 1948 he was studying continuously, and in November he completed his leaving certificate requirements. By February, 1949, activity had subsided, and it was decided to arthrodese his sacro-iliac joint. Unfortunately that had to be postponed owing to folliculitis around the buttocks, but it was eventually performed in April, 1949. Bone chips were taken from his iliac crest, and packed into a gutter across the joint. He remained in a plaster bed for another three months and was then fitted with corsets and allowed to get up. He was discharged from hospital in September, 1949, wearing his corsets. He was well until April, 1951, when he noticed a slightly tender lump, which was gradually increasing in size, on the outer side of his right hip. He was readmitted to hospital, and in May, 1951, a bursa over the greater trochanter was found to contain caseous material; a small amount of pus was removed, from which tubercle bacilli were grown. Recovery was uneventful, and he was discharged from hospital two weeks later. Since then he had remained quite well.

Tuberculosis of Right Hip Joint.

Dr. Hamilton showed a man, aged twenty-three years, a truck driver, who had "dislocated" his right hip in January, 1946. A few months after recovery he had noticed that his hip was again painful and movement in it limited. He received hospital treatment, with alleviation of symptoms except for a limp, which persisted up to the time of his admission to the Royal North Shore Hospital of Sydney. He had arrived in Australia from Yugoslavia in April, 1949. Ten months before the time of the meeting his limping had increased, and after seeking medical advice he was placed in a plaster spica for six weeks, followed by massage, all with little improvement. When admitted to the Royal North Shore Hospital of Sydney on February 1, 1952, he had complained of pain on walking, stiffness in the right hip, especially in the morning, occasional night sweats and loss of weight. Physical examination revealed that he was an apparently healthy young man with flexion deformity of 30° and limitation of all movements in the right hip. Enlarged firm glands were found in the right femoral triangle, and considerable wasting of the quadriceps was noted. X-ray examination of the right hip suggested a tuberculous process with soft tissue swelling below it. The chest was radiologically normal. The result of a full blood count was within normal limits, and the blood sedimentation rate was ten millimetres per hour. The patient was placed on a Jones's abduction frame with fixed traction two weeks after admission to hospital. Immobilization was maintained, and one month later aspiration of the swelling below the joint was performed. Tubercle bacilli were present in 80 millilitres of fluid withdrawn. Further aspiration was carried out on March 31, 1952, when 120 millilitres of a dark straw-coloured fluid were withdrawn.

Old Tuberculosis of Left Hip and Bone Graft.

Dr. Hamilton's next patient, a single woman, aged thirty-four years, had twenty years before been diagnosed as suffering from tuberculosis of the left hip. Ten years later an operation was performed to correct adduction and flexion deformity. The patient was then well until twelve months before the time of the meeting, when increasing pain and difficulty in walking were noticed, together with shortening of the left leg. She was admitted to hospital on February 7, 1952, when she appeared to be in good general health. Thoracic scoliosis was present, and the left thigh was flexed, adducted and internally rotated with gross limitation of all movements. No other relevant abnormality was found. After preliminary X-ray examination (which revealed long-standing tuberculous disease of the hip and evidence of a previous osteotomy), the following surgical treatment was carried out: (i) bone graft of the great trochanter and neck of the femur to the ilium; (ii) osteotomy of the upper end of the femur to correct flexion and adduction deformity; (iii) immobilization on Jones's frame with the left leg abducted to 30°. The patient was progressing satisfactorily.

Tuberculosis of Spine and Left Wrist.

Dr. Hamilton showed a single woman, aged thirty-four years, who two years prior to admission to hospital had noticed pain across the back between the shoulder blades. The pain gradually increased in frequency and severity and was almost constant when she was admitted to hospital in October, 1950. The patient also noticed that her left wrist had been swollen over the same period. Examination of the patient revealed a thin woman in no apparent distress with a non-fluctuant lump extending between the eighth and twelfth thoracic vertebræ posteriorly. No limitation of spinal movement was observed. X-ray examination showed tuberculous disease affecting the seventh and eighth thoracic vertebræ, and, in addition, the left triquetral bone was similarly affected. The patient was nursed on a Jones's frame, and plaster of Paris was applied to her left forearm and wrist. Eleven months later a bone graft was performed on the affected portion of the spine, the donor sites being both posterior iliac crests. The patient was then kept at

rest in a plaster bed. Three months later, in December, 1951, she was allowed up in a spinal brace, while her wrist was immobilized in a Crabbe splint. It was found that the patient also had renal tuberculosis, though excretion pyelography showed no positive signs in either kidney. On the advice of the honorary urologist nothing further had been done about the renal condition up to date. The patient had been discharged from hospital in December, 1951, wearing a spinal brace.

Tuberculosis of the Spine.

Dr. Hamilton then showed a single woman, aged twenty-one years, a Lithuanian, who had arrived in Australia in May, 1948. In December of the same year she had an attack of dry pleurisy, from which she made an apparent recovery. In February, 1949, she developed a severe lumbar backache, which, after four weeks, subsided without treatment. However, in September, 1949, the backache returned, this time accompanied by high fever. She was admitted to Prince Henry Hospital, but left two weeks later, against advice, without a diagnosis having been established. One month later she was admitted to the Royal North Shore Hospital of Sydney with a diagnosis of miliary tuberculosis and tuberculous disease of the spine. She received a course of streptomycin (three months), after which examination of the lung fields showed no active lesion. During the course of streptomycin she was placed on a frame (that was in January, 1950), and seven months later she was discharged from hospital wearing a spinal brace. In January, 1951, the patient noticed a swelling in the upper posterior aspect of the right buttock. However, on examination a firm mass was detected in the right iliac fossa, a diagnosis of psoas abscess was made, and she was readmitted to hospital in February, 1951. X-ray examination showed tuberculous involvement of adjacent borders of the eleventh and twelfth thoracic vertebræ. The chest was radiologically normal. The patient was placed on a Jones's frame, and over the last thirteen months there had been a steady improvement in the X-ray appearances of the involved vertebræ, although there was still some activity present. The abscess gradually subsided, and was scarcely palpable four months after the patient's admission to hospital. She was quite symptomless at the time of the meeting.

Tuberculosis of the Spine and Psoas Abscess.

Dr. Hamilton, in conjunction with DR. L. MACDONALD and DR. C. D. LANGTON, showed a man, aged twenty-two years, a sawmiller, who had arrived in Australia in October, 1948. He was admitted to hospital on September 1, 1951, complaining of pain in the right hip radiating to the knee for four months, lack of appetite and loss of weight. He had had a biopsy performed on his right foot in April, 1951, and tuberculosis had been diagnosed. On admission to hospital he was tender in the right groin, his right inguinal lymph glands were enlarged, and all hip movements were restricted. He was also tender over the right sacro-iliac joint. There was a short leg plaster on his right leg. The plaster was removed, and skin traction with a club foot shoe was applied, while he was waiting for his Jones's frame. X-ray examination on admission to hospital showed no abnormality in the chest or right hip, while on September 20 a destructive lesion of the third lumbar vertebra and narrowing of the third and fourth lumbar disks were observed. The bones of the right foot were decalcified, and the scapho-cuneiform joints were irregular. Two weeks later his temperature rose, he complained of continuous headache and felt generally unwell, and several days later he became drowsy and uncommunicative. Lumbar puncture was performed, but examination of the cerebro-spinal fluid revealed no abnormality. On September 22 he developed a swelling above and below the inguinal ligament, and a psoas abscess was diagnosed. Several days later he was placed on a Jones's double abduction frame, on which he had remained ever since. The swelling increased in size, and was aspirated on December 21, 300 millilitres of pus being withdrawn. Tubercle bacilli were grown from the pus. He also developed a swelling on the inner side of the right buttock, from which tuberculous pus was aspirated. On January 19, 1952, administration of streptomycin, 0·5 gramme twice in the day every second day, together with PAS, 10 grammes every second day, was begun and had been continued ever since. Throughout the early months of 1952 his condition had been fair; periodic attacks of headache and vomiting had resulted in further lumbar puncture being performed, but on no occasion were abnormalities detected in the cerebro-spinal fluid. X-ray examinations in February, 1952, showed no further bone destruction in the lumbar part of the spine and a little more sclerosis around the affected joints in the right foot.

Tuberculous Right Knee.

Dr. Hamilton, Dr. Macdonald and Dr. Langton also showed a man, aged twenty-four years, a plasterer, who had arrived in Australia in May, 1950. Eighteen months prior to his admission to hospital in April, 1951, the patient had noticed a painless swelling of the right knee. It caused him little discomfort until two months before admission to hospital when it increased greatly in size and became painful. On his admission to hospital there was an obvious swelling of the right knee joint, which was abnormally mobile. A "patellar tap" sign was present; pain on full flexion and wasting of the quadriceps were noted. Investigations showed negative findings from Wassermann and gonococcal fixation tests and a blood sedimentation rate of eight millimetres in one hour, and X-ray examination revealed rarefaction of the bones around the knee joint. A prostatic smear examination also yielded negative findings. Diagnosis was in doubt until September 28, when biopsy of two enlarged inguinal lymph glands showed tuberculosis. The patient was given 1,000,000 units of penicillin a day for four weeks after admission to hospital; on September 29 streptomycin administration was begun, 0·5 gramme being given by intramuscular injection twice daily for four months, a total of 110 grammes in all. Simultaneously PAS, 10 grammes daily in four divided doses, was administered throughout. In addition 0·5 gramme of streptomycin was injected into his knee joint thrice weekly for a total of 20 injections. He was placed in a Thomas splint on October 1 and remained in it until March, 1952, when a walking caliper was applied. His general condition had remained good throughout.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

FROM ALEXR. McLEAY TO THE SURGEON SUPERINTENDENT OF THE SHIP AMERICA.[1]

Colonial Secretary's Office,
21 August, 1829.

Alexr. Stewart, Esq.,
Surgeon Superintendent,
Ship America.
Sir,

In acknowledging the receipt of your letter of the 20th instant, reporting the death of John HUMPHRIES[2] and enquiring in what way the Body is to be disposed of, I am directed by His Excellency the Governor to request that you will cause it to be taken to some distance outside the Heads of Port Jackson and there to be Committed to the Deep care being taken that there is a sufficient weight attached to it to sink it.

I have, &c.,
ALEXR. McLEAY.

Correspondence.

PENICILLIN INJECTIONS IN DOMICILIARY VISITS.

SIR: Many practitioners have found that the giving of intramuscular injections of penicillin in private houses leads to much waste of time in sterilizing equipment. Needles and syringes cannot be used from antiseptic storage because of inactivation of antibiotic substances; wax and oily solutions also require a dry needle.

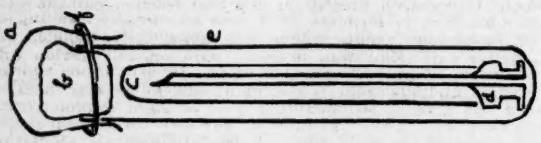
I have found that the disposable plastic syringe together with specially prepared needles obviates all this trouble. The parts needed are as follows: a glass tube three inches by one-half inch (Wassermann tube); a metal sheath two

[1] From the original in the Mitchell Library, Sydney.

[2] The ship *America* was in quarantine at the time of this man's death owing to an epidemic of measles and the prevalence of dysentery amongst the personnel on board.

and three-eighth inches (made from bottom portion of an old thermometer case); a needle 19G by two inches; cotton wool, a rubber band and a paper cap. They are assembled as shown in the accompanying diagram.

By courtesy of the theatre sister I have had these autoclaved at the local hospital in batches of one dozen.



(*a*) Paper cap. (*b*) Cotton wool. (*c*) Metal sheath. (*d*) Needle. (*e*) Glass tube. (*f*) Rubber band.

To use, the cap and cotton plug are removed, the glass tube is inverted and the needle in its sheath is allowed to slide out. The sheath is then held in one hand while the syrette nozzle is pushed into the needle mount with the other.

The injection can be given in less than one minute in perfect sterility. All parts can be used repeatedly after cleaning.

Yours, etc.,
B. D. VAUGHAN.

5 Tyers Street,
Portland,
Victoria.
July 24, 1952.

SINGAPORE PÆDIATRIC SOCIETY.

SIR: The following information may be of interest to you. On August 1 the Singapore Pædiatric Society was formed to advance the knowledge and practice of pædiatrics and to promote child health. This is the first pædiatric society to be formed in Malaya, and is open to all doctors registered in the Colony of Singapore or in the Federation of Malaya.

The office-bearers are as follows: *President*: Dr. G. Haridas, Pædiatric Specialist, Colony of Singapore. *Vice-President*: Professor E. S. Monterio, Professor of Clinical Medicine, University of Malaya. *Honorary Treasurer*: Dr. William Heng. *Honorary Secretary*: Dr. G. Keys Smith, Medical Officer-in-Charge, Saint Andrew's Mission Hospital for Children, Singapore. *Elected Members of the Executive Committee*: Professor J. A. P. Cameron, Professor of Orthopædic Surgery, University of Malaya; Dr. Tan Jiak Hoon, Infant Welfare Department, Singapore; Dr. W. B. Young, Radiologist, General Hospital, Singapore; Dr. Wong Kin Yip, Ophthalmologist, General Hospital, Singapore.

Yours, etc.,
G. KEYS SMITH.

Saint Andrew's Mission Hospital,
Singapore,
August 14, 1952.

UNITED STATES INFORMATION LIBRARIES.

SIR: We have been informed by the United States Information Library in Melbourne that several inquiries have been received recently from physicians regarding the availability of the "American Medical Directory". This directory may be found in both the Sydney and the Melbourne United States Information Libraries.

There are other titles which may be of interest as well, for example, "Current Therapy, 1951", "The Year Book of Dermatology and Syphilology, 1951", "The Year Book of Drug Therapy, 1951", "The Year Book of General Surgery, 1951", "The Year Book of Medicine, 1951", "The Year Book of Neurology, Psychiatry and Neurosurgery, 1951", "The Year Book of Obstetrics and Gynecology, 1951", "The Year Book of Orthopedics and Traumatic Surgery, 1951", "The Year Book of Pediatrics, 1951", "The Year Book of Radiology, 1951", "The Year Book of the Eye, Ear, Nose and Throat, 1951", "The Year Book of Urology, 1951". Both libraries subscribe to *The Journal of the American Medical Association* as well. The libraries are located as follows: care of the Public Library of New South Wales, Macquarie Street, Sydney; care of the Public Library of Victoria, corner of Swanston and Latrobe Streets, Melbourne.

We should be happy to have any of your readers visit our library, and see what is available in our medical section.

Yours, etc.,
MARY LOU BOWERS,
Director, U.S.I.S. Library.

C.o. Public Library of New South Wales,
Macquarie Street, Sydney,
August 15, 1952.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Tamworth.

THE Post-Graduate Committee in Medicine, in conjunction with the Northern District Medical Association, will hold a week-end course at Tamworth on Saturday and Sunday, September 13 and 14, 1952. The programme will be as follows:

Saturday, September 13: 2 p.m., meeting of the Northern District Medical Association.

Sunday, September 14, at the Tamworth Base Hospital: 9.30 a.m., "The Use of Anticoagulants in Coronary Disease", Dr. J. Halliday; 11 a.m., "Recognition and Treatment of Bronchiectasis and Empyema", Dr. H. Windsor; 2 p.m., "Some Drugs—Old and New", Dr. J. Halliday; 3.30 p.m., "Recognition and Treatment of Neoplasm of the Lung", Dr. H. Windsor.

The fee for attendance at the course will be £3 3s. Those wishing to attend are requested to notify Dr. G. Archbold, Honorary Secretary, Northern District Medical Association, 67 Bridge Street, West Tamworth, as soon as possible.

Clinical Meeting at Balmoral Naval Hospital.

The Post-Graduate Committee in Medicine in the University of Sydney announces that a clinical meeting will be held at the Balmoral Naval Hospital on Tuesday, September 9, 1952, at 2 p.m., when Dr. J. Cobley will speak on "The Diagnosis and Management of a Diabetic with Some Discussion on Complications". Clinical cases will be shown after the lecture. All members of the medical profession are cordially invited to attend.

NUFFIELD FOUNDATION DOMINION TRAVELLING FELLOWSHIPS.

THE Chairman of the Nuffield Foundation Australian Advisory Committee (Sir John Medley) has announced that the Foundation will offer to Australian graduates seven travelling Fellowships to begin in 1953. Three of these will be in medicine, the others being in the natural sciences (2), the humanities (1) and the social sciences (1).

The purpose of the Fellowships is to enable men or women who are Australian graduates of outstanding ability to gain experience and training in the United Kingdom in their chosen fields, and to make contact there with scholars working in those fields, with a view to the Fellows' equipping themselves to take up senior posts in research and teaching in Australia. A Fellow will be expected to resume residence in Australia on the completion of the Fellowship.

The awards are available to Australian nationals, normally between the ages of twenty-five and thirty-five years, preferably holding a master's or doctor's degree, with a year or more of subsequent teaching or research experience on the staff of a university or comparable institution.

The Fellowships will normally be tenable for one year. Their value will vary with individual circumstances, but will in no case be less than £900 sterling *per annum*. The Foundation will in appropriate cases meet the travelling expenses of a Fellow's wife.

Applications for 1953 Fellowships should be submitted not later than November 1, 1952, to the Secretary, Nuffield Foundation Australian Advisory Committee, c.o. The Australian National University, Canberra, A.C.T. As the Secretary will not be back from England until the middle of September, application forms should be obtained from the Acting Secretary, c.o. Vice-Chancellor's Office, University of Melbourne, Carlton, N.3, Victoria.

Obituary.

HUGH WILLIAM BELL CAIRNS.

DR. LEONARD C. E. LINDON has sent the following appreciation of the late Sir Hugh William Bell Cairns.

Hugh William Bell Cairns died in Oxford on July 18 at the age of fifty-six years, leaving behind him a record of achievement which has not been equalled by any graduate of the Adelaide Medical School. At his birth he was blessed, not by worldly goods, but by an enormous capacity for work and a determination to learn, qualities which were inherited from his Scottish forebears. He also inherited magnificent physical fitness, very necessary to him in the performance of frequent and exhausting craniotomies.

Born at Riverton, a country town in South Australia, his early school days were spent at Riverton High School; from this beginning, scholarships took him through the whole of his subsequent academic education at Adelaide High School, the University of Adelaide and the University of Oxford. His medical course was interrupted by the First World War, when he served as a private in the Third Australian General Hospital on Lemnos; he was then recalled, to complete his medical course, which he did at the end of 1917, and returned to France to serve as a captain in the Australian Army Medical Corps throughout 1918. He was elected the 1917 Rhodes Scholar for South Australia, but did not go up to Oxford until 1919. While at Oxford he came under the influence of two great men, Sir Charles Sherrington and the late A. L. Smith, Master of Balliol. By the former, he was inspired to continue research into the physiology of the central nervous system; and from the latter he acquired the love of Oxford, her traditions and her culture, factors which in later years helped him in his decision to leave his private practice in London, and return to Oxford to work, to teach, and to organize a research neurosurgical clinic at the request of Lord Nuffield.

In November, 1921, he gained his Fellowship of the Royal College of Surgeons, and shortly afterwards married Barbara, the youngest daughter of the Master of Balliol. Then followed four hard, lean years. Cairns was determined to get on the staff of the London Hospital; and these years of waiting were occupied by coaching and by working as an assistant in the department of pathology and the out-patient department. These were indeed hard though fruitful years; but in 1926 his appointment as assistant honorary surgeon to the London Hospital marked the turning of the tide. A few months later, a Rockefeller Fellowship took him to Boston, where he worked for a year as one of the team of Harvey Cushing; and Cairns's career as a neurosurgeon had really begun. In later years Cushing frequently manifested his admiration for Cairns and his high opinion of his ability; and Cairns's friendship was the best introduction that any visitor to Cushing's clinic could have. On his return to London, the staff of the London Hospital, by an extremely wise and generous action, encouraged him in the organization and development of a neurosurgical clinic. The visitor to Great Britain today takes the existence of innumerable well-organized clinics for granted. But twenty-five years ago they did not exist. And it stands greatly to his credit that Cairns was able to impress upon his colleagues the reason for such a clinic, and his ability to organize and to justify it. From its very inception, he began to produce results the like of which had not been seen in London, and for the remaining twenty-five years of his life he was overwhelmed with work. In 1936 he was offered the Nuffield Chair of Surgery in the University of Oxford; with very little hesitation he sacrificed the certainty of a very lucrative practice in London for the attractions of life in a department of surgical research in Oxford. Lord Nuffield knew his man; not only did Cairns make his clinic the outstanding neurosurgical research centre in England, but he built up the whole department of general surgery. He worked his assistants as hard as he worked himself, but he trained them. And many of our Australian neurosurgeons have to thank Hugh Cairns for his inspiring teaching and help.

In the Second World War he was Consulting Neurosurgeon to the British Army, and to the civilian emergency medical service; he made great contributions to the treatment of head injuries, the establishment of head injury centres and the treatment of intracranial suppuration. For these services he was created a Knight Commander of the Most Excellent Order of the British Empire in 1946. His team of co-workers was largely responsible for the clinical application of penicillin, after its discovery by Fleming and Florey. In 1948, after an absence of thirty years, he returned to Australia as the first Sims Commonwealth Travelling Professor, an appointment which gave to many of his old fellow students the great pleasure of meeting him again.

His energy seemed inexhaustible. In his later years, much time was spent in visiting European and American clinics to give lectures and addresses. And space does not permit a record of the many neurosurgical bodies which honoured him with membership, nor of the volume of important work which has been published under his direction by the members of his clinic. He received many honorary degrees and was for many years a member of the Council of the Royal College of Surgeons.

At work, Hugh Cairns was silent, intense and patient, and demanded the same high standard of performance from the whole team as from himself. From Cushing he had inherited the faculty of analysing the causes of failure, and of working out the best method whereby to avoid repetition of failures. And the clarity and detail of his case records were a masterpiece. Away from work, he was a man of great charm and sense of humour, with an interest in all things. He rowed for Adelaide in 1914, and for Oxford in 1920; but also played good games at lacrosse and tennis. The writer acted as his caddie on the links at Stockholm, and was pleased to find at least one chink in Cairns's armour. In later years, he loved long walks in the country; and to accompany him on these rambles was to enjoy discussions on the widest range of subjects. To visit him and Lady Cairns, at intervals of many years, was to find a most happy couple who had remained young and entirely unchanged by the honours which had fallen upon them. To her, and to her four children, we offer the deepest sympathy and regret in the untimely loss of a really great man. As in the case of his old "Chief" at Boston, his monument shall be the record of his achievements and the inspiration he gave to a succession of young neurosurgeons throughout the Empire.

Dr. Douglas Miller writes: Hugh Cairns was a very remarkable man. His achievement in life was compounded of much more than professional skill and knowledge. He had an almost uncanny faculty for seeing what he would call "a good line" in work or investigation. It was this ability which in early years brought him to realize that the school of Harvey Cushing had much to offer to the field of neurological surgery in England. London was supreme in clinical neurological research and teaching, but the surgical method had not changed since the days of Horsley, and the results were discreditable.

So it was that Cairns left London and worked with Cushing. He took from his great master a pattern of work and life, to which he adhered with religious observance.

His devotion to neurosurgical problems and his capacity for following a clue or a desperate hope to its utmost length very soon bore fruit. While yet in a most junior position at the London Hospital he was marked as a forerunner in the revolution of neurological surgery.

He was completely absorbed by the scientific and practical aspects of this work. His forceful personality enabled him to arrange his life in what he considered the best possible way for success. At the London Hospital and later at Oxford, he won the right to treat all classes of patients in the hospital. This ideal must have cost him financial sacrifice and also exposed him to professional criticism, to both of which he would be peculiarly impervious if he considered his action right. It did, however, enable him to spend his whole days in the centre of his problems.

He once told me with pride that he had never had a "brass plate". There is no doubt that if he had had such a symbol in Harley Street, distracting his attention from the hospital, neurological surgery would have suffered.

Reference to the literature of almost any neurosurgical subject will show how much we gained by his persistence in his single-minded method. He was yet very young when he attracted men from overseas to work under his direction. A little later other learners came from European centres, and today his pupils are to be found all over the world. Many owe to Cairns much more than a knowledge of their craft, if they became touched by his devotion and unqualified dedication to what he called a great and dignified work; though working with him was not always easy or pleasant.

He was, at first, a somewhat dour personality, and the outside defences against familiarity or friendliness were hard to penetrate. When one did get to know him, the reward was great. He had a crystal-clear mind and a totally dedicated heart. He had a most extraordinary facility for rapidly and accurately assessing a person, or a situation, and no doubt this strength brought him many tactical victories. His criticism of men and methods was fearless and direct, usually expressed picturesquely and in a few words. His faculty for self-criticism was just as incisive.

I remember once, after going through an elaborate research institution, where equipment was in excess of work or workers, he remarked: "It reminds me of the *Marie Celeste*." On another occasion, after watching a surgeon rather fumble a technical operative procedure, he said: "I learned at that operation that if you get lost it might be a good idea to ask an onlooker the way."

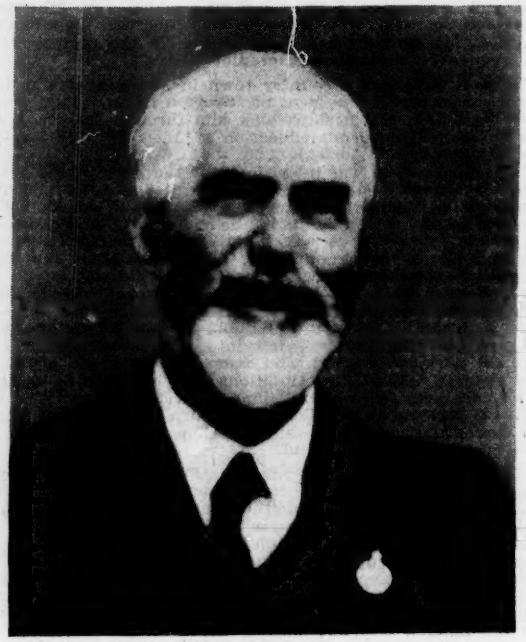
In equal measure his appreciation or praise were sincere and most valued, and there are many who will retain a proud memory of the kindly encouragement, intense look and winning smile, which meant that they had satisfied their revered "H.C.".

FREDERICK GUY GRIFFITHS.

DR. DOUGLAS ANDERSON writes: Dr. Guy Griffiths, whose death was reported in the journal recently, was for many years one of the most outstanding figures amongst the medical profession in Sydney.

He was the son of George Neville Griffiths, who was a grazier with property in New South Wales and Queensland, and he was born on July 18, 1876. He received his school education at the Cooerwull Academy and for the last two years at the Sydney Grammar School, where he won the senior Knox Prize for general proficiency. He matriculated in 1895, gaining the Aitken Scholarship for general proficiency and the Barker Scholarship Number Two for mathematics.

At the university he first entered the faculty of arts and in 1896 won the George Allen Scholarship, another mathematical award. In 1897 he entered the faculty of medicine, but continued his mathematical studies as an evening student, winning the Barker Scholarship Number One in that year and graduating as bachelor of arts with honours in 1898. He was a resident at Saint Andrew's college, and there, too, he annexed various scholarships and prizes as well as representing the college in many intercollegiate contests—rowing, running, cricket, lawn tennis and football—and acting as college secretary. According to a writer in *Hermes* he was "a man of no mean experience and prowess in training Freshmen in the way they should go". In the wider field of university sport he was almost as versatile: he rowed in three different crews in maiden and junior events, in athletics he won the mile race, and at lawn tennis he played with great success for many years. He took a leading part in the activities of all sorts of university clubs and societies: he was a formidable debater and became honorary secretary of the Sydney University Union in 1896; he served on the editorial committee of *Hermes* and on the committee of the Boat Club; he was treasurer of the Athletic Club for two years and on the committee of the Undergraduates' Association for several years, becoming President in 1899. He was in successive years treasurer and



secretary of the Medical Society and in 1901 he was President. He graduated as bachelor of medicine in 1900. In 1911 he was admitted to the degree of doctor of medicine, his thesis being on the use of tuberculin in diagnosis and treatment, and in the same year he took the degree of master of surgery. His brilliant university career was still not finished, for in 1948 his lifelong interest in mathematics led him to proceed in that subject to the degree of master of arts.

Dr. Griffiths was a resident medical officer at Sydney Hospital in 1901, and in the following year he served as resident pathologist under Sydney Jamieson. After leaving the hospital he went into general practice at Gundagai for a while, after which in 1904 he commenced practice as a physician in Macquarie Street. For forty-five years he served on the honorary medical staff of the Royal North Shore Hospital of Sydney—he was honorary assistant pathologist from 1905 to 1909, honorary pathologist from 1909 to 1914, honorary physician from 1914 to 1938, and honorary consulting physician from 1940 till his death. He delivered the nurses' medical lectures at the hospital for twenty-five years. He was also for many years honorary physician to the Anti-Tuberculosis Association of New South Wales and a member of the Special Tuberculosis Board of the Repatriation Commission of Australia. In 1938 he became one of the foundation Fellows of The Royal Australasian College of Physicians.

In 1917 Dr. Griffiths enlisted in the Australian Imperial Force. He served as physician and pathologist to the Second Australian Stationary Hospital, Moascar, Ismailia and Lines of Communication from October, 1917, to March, 1919, and as physician to the Fourteenth Australian General Hospital, Port Said, with the rank of major from April to July, 1919. He was mentioned in dispatches. In the Second World War he served with the Volunteer Defence Corps from 1939 to 1946, at first as a private, later as a medical officer with the rank of captain.

Dr. Griffiths was for many years a member of the Union Club, where he indulged a predilection for the game of bridge, at which with his mathematical mind he was most adept. In 1902 he was married to Miss Elizabeth Deane, who survives him with two sons and three daughters.

I first met Dr. Griffiths when I came to the Royal North Shore Hospital as a resident medical officer twenty years ago. He was tall and upright in carriage and possessed all the gravity, geniality and assurance that any situation might require. His moustache and beard, like Robert Koch's, and a faint suggestion of individuality in his hats and clothes, which I shall mention later, enhanced his very striking presence. His red lips and a something in his eye indicated his enjoyment of life. His speech was very clear and unhurried and his diction precise as befitted the clarity of his mind; in fact, I should say that he was one of the best orators in Sydney, though he was apt to cultivate the orotundity of fifty years ago.

At medical meetings he would always sit in the front seats. If it were possible he would first open the windows: he always affected incredulity that any medical man could imagine harm in "draughts" or "night air". At annual general meetings no one could ask an awkward question with more aplomb.

Dr. Griffiths was the personification of courtesy, often referred to nowadays as old-world courtesy. This was apparent in all his conversations and actions. He was certainly one who "never gave offence unintentionally". One of his old-world mannerisms was a little bow, a slight, quick, momentarily sustained inclination, which everyone associated with him. I remember one resident medical officer at the Royal North Shore Hospital who was transferred from Dr. Griffiths to a gynæcologist who was O.C. miscarriages: "Dr. Griffiths has taught me to bow, sir", he said to his new chief at their first meeting; "I hope you will teach me to scrape."

At that time Dr. Griffiths was already the senior physician. He was about fifty-five years of age, but he looked over sixty: however, in the next twenty years he seemed to age very little. He possessed a wealth of reminiscence of earlier days, methods and personalities. He was fond of recalling that for many years he undertook all examinations of the blood—hæmoglobin determinations, corpuscle counts, microscopical examinations and the like—on the rare occasions when these were thought necessary. Curiously he was lukewarm when the pathology department was brought into being at North Shore, and he made use of it sparingly, though this may have indicated a wisdom which we just at this present day do not appreciate. His chief medical interest was tuberculosis—"Studies in Tuberculosis" had been the title of his thesis for the doctorate. He was an enthusiastic tuberculin therapist (being a disciple of William Camac Wilkinson), and in 1914 he founded at the hospital a Tuberculin Dispensary which functioned on Wednesday afternoons, but he did not himself conduct it after his return from the war. It continued for about ten years, after which, with the appointment of a specialist physician to take charge of it, tuberculin disappeared, and it was renamed the Anti-Tuberculosis Dispensary and later the Clinic for Pulmonary Diseases. Dr. Griffiths continued to treat tuberculosis patients in his own beds with tuberculin and used to bring it to the hospital from his rooms, each individual dose in a tiny rubber-stoppered bottle wrapped in a page of an old telephone directory.

Dr. Griffiths represented the honorary medical staff of the hospital on the board of directors for many years and was a member of the house committee. In those days two members of the house committee, of whom Dr. Griffiths was one, visited the hospital every Sunday and made friendly conversation with the patients, the nursing staff and the lay workers. By these visits he kept his finger on the pulse of the hospital, and also, I have been told, inspired confidence and trust. He was generally regarded as a most fair-minded man. Just before the trouble in 1938 which led to changes in the administration of the hospital, Dr. Griffiths was chairman of the honorary medical staff and I, a very junior member, was secretary. The Board of the day invited applications for appointment to positions on the staff and ominously withheld the usual sentence about those already holding the positions being eligible for reappointment. The staff resolved to send a letter to THE MEDICAL JOURNAL OF AUSTRALIA making it clear that all holders of the positions were eligible for reappointment and that they would presumably apply. I duly wrote the letter, but Dr. Griffiths requested me to allow him to sign and deliver it, making the remark, of which I did not grasp the full significance at the time, that it was a "two-edged letter". It was published in the

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 2, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	1	..	..	..	..	..	..	1
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1(1)	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	6(6)	..	2	..	1	..	..	9
Diphtheria	11(1)	1(1)	3(2)	1(1)	1(1)	..	..	..	17
Dysentery (Bacillary)	..	1(1)	4(1)	..	..	..	..	..	5
Encephalitis	..	2(1)	..	..	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	..	..	..	..	17(11)	..	..	..	17
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	3	..	3
Leptospirosis	..	..	1(1)	..	..	..	..	..	1
Malaria	..	..	1(1)	..	..	..	1	..	2
Meningococcal Infection	5(3)	3(2)	1(1)	1(1)	1(1)	2(2)	..	..	13
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	5(3)	3(2)	1	19(8)	1	..	..	..	29
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	40(11)	..	..	..	..	..	..	40
Salmonella Infection	..	..	..	..	1(1)	..	..	..	1
Scarlet Fever	24(16)	29(2)	14(10)	15(3)	3(3)	11(3)	..	..	96
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	1	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	29(23)	4(2)	8(2)	10(8)	22(9)	7(2)	1	..	81
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	2(1)	..	2(1)	..	..	..	4
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

journal and exasperated the Board. In revenge they deprived Dr. Griffiths of his own appointment, a possibility which he had undoubtedly foreseen when he prevented me from signing the letter.

The jiggery-pokery with the staff appointments aroused violent emotions at the time, but everyone was dumbfounded at the supersession of Dr. Griffiths. Some said it would break his heart and kill him. "Old Guy" did not utter a word of criticism or reproach, and if I have any knowledge of him he did not lose a wink of sleep. His philosophy was Olympian and his hospital service anachronistically altruistic. Dr. Griffiths was happily restored to the consulting staff when the present Board took office in 1940, and he often appeared at hospital gatherings. It would be obvious that all the old-timers were delighted to see him, and his manifest dignity and prestige must have helped to impress upon newcomers that the old "back numbers" were men of stature and would take some living up to.

Dr. Griffiths, when he entered the hospital, always used to bow to a marble tablet in the front hall upon which the name of his brother, killed at Lone Pine, was inscribed in a list of benefactors. This semi-weekly rite often surprised those who witnessed it for the first time. He affected a contempt for persons, including his colleagues, who had performed no military service, and on occasions he expressed this in various ways with startling openness.

I was Dr. Griffiths's resident at the Royal North Shore Hospital for a couple of terms. I used to think that he was a very good physician in his knowledge and professional competence and the equal of any of my undergraduate teachers. His punctuality for rounds, his punctilio in matters of etiquette, his considerateness towards patients and his ripe "bedside manner" must have been good for his residents to experience though they might not emulate them. I remember well his gravity with children and how well his serious manner and readiness to explain matters truthfully without talking down seemed to succeed with them.

I have mentioned Dr. Griffiths's slight departure from convention in his attire. These were not dictated by whimsy. For years he showed by example, which, of course, no one ever followed, how men's dress might be made more comfortable and healthy with but little variation from the fashion. He never had turn-up trouser cuffs to collect debris, his shirts were open-wove cotton, and his felt hats were worn without the usual depression in the crown. One day he left his hat over in the isolation block of the hospital, and his resident offered to go back for it. On the way back the resident foolishly placed a fashionable dent in it. "Old Grif" eyed the proffered headgear but did not accept it. "My hat had no deformity of the crown", he said, "would you be so kind as to go back and see if you can find such a hat?" The resident went back, his mission was successful, and all faces were saved.

I knew little of Dr. Griffiths except for my association with him at the Royal North Shore Hospital, and consequently the personal part of this memoir reflects only one facet of his colourful and many-sided personality. Perhaps also it gives undue prominence to his little eccentricities, which eccentricities, however, he carried like a banner. I hope it is sufficient to give some inkling of the solid worth of his service to the hospital and to recall that when he represented his colleagues their dignity and wider interests were in the safest of hands.

ROBERT JOSEPH TAYLOR.

We regret to announce the death of Dr. Robert Joseph Taylor, which occurred on August 19, 1952, at Sydney.

Medical Appointments.

Dr. S. B. Sutton has been appointed a member of the Panel of Official Visitors to the Mental Hospital and Receiving House, Royal Park, Victoria.

Dr. D. M. Anderson has been appointed medical superintendent, Lachlan Park Hospital, Department of Public Health, Tasmania.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Howell, David, M.B., B.S., 1950 (Univ. Sydney), Royal Hospital for Women, Paddington, New South Wales.

Diary for the Month.

SEPT. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 3.—Western Australian Branch, B.M.A.: Council Meeting.
SEPT. 5.—Queensland Branch, B.M.A.: Jackson Lecture.
SEPT. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 12.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 15.—Victorian Branch, B.M.A.: Finance Subcommittee.
SEPT. 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital: all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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